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# Review of Neurology and Psychiatry

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## Original Articles

### **A CONTRIBUTION TO THE STUDY OF SECONDARY DESCENDING DEGENERATION IN THE POSTERIOR COLUMNS OF THE SPINAL CORD.**

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SINCE Türck in 1851 first traced degenerated nerve fibres from the brain into the spinal cord, an enormous amount of work has been done on that organ which has enlarged our knowledge of its structure and functions. In this country, Bastian, Ferrier, Gowers, Horsley, Hughlings Jackson, Mott, Sherrington, Schäfer and others have devoted a large amount of valuable time and thought to this subject; whilst abroad, Erb, Edinger, van Gehuchten, Cajal, Obersteiner, Charcot, Déjerine, Pierre Marie, Retzius and a number of others have also given with success a great deal of valuable time to the same purpose. With the advent of fresh discoveries, it has been shown that the nervous system is inconceivably complex, and that systems, tracts, or nerves which formerly seemed simple units, have been analysed into more elaborate details, whilst new structures with necessarily diverse functions have been found as constituent parts of, or in association with, what was formerly thought to be some simple organ. Amongst many instances that could be cited this applies with especial force to the view formerly held of the structure of the posterior columns of the spinal cord which, at

one stage of our knowledge, were supposed to possess merely nerve fibres which, when they underwent secondary degeneration, always did so in an ascending direction (*i.e.* on the cerebral side of the lesion producing the degeneration). It was also supposed that the posterior columns only conveyed afferent impulses (*i.e.* nervous impulses, passing upwards towards the brain).

The first to show that this supposed simple arrangement was not the true one was Dr Charlton Bastian (1), who as early as 1867 figured a small descending tract in the posterior columns of the spinal cord which had degenerated in man after an injury to the cervical region.<sup>1</sup>

About 1876, Flechsig (2) showed by the embryological method, of which he has been so great an exponent, that a small area named by him the oval field could be mapped out on either side of the middle line in the lumbar region of the spinal cord, and that these fibres degenerated in the opposite direction to those around them. In 1880, Kahler and Pick (3), Strümpell (4), and Westphal (5), all published cases which, as a result of compression more or less complete at various levels of the spinal cord, produced descending secondary degeneration in the posterior columns; and three years later, Schültze (6) published similar cases showing the same appearances, and particularly pointed out the presence of a comma-shaped tract, which degenerated downwards as a result of compression of the spinal cord in the cervical region. This tract, larger anteriorly, more pointed posteriorly, he described as extending in the posterior external columns for a distance downwards of two and a half centimetres. But as will be seen below, the tract is much more extensive than Schültze at that time supposed.

In 1889, Howard Tooth (7) found the so-called comma tract (in a case of spinal compression) degenerated from the eighth cervical segment down to the eighth dorsal; and in another case from the seventh cervical to the seventh dorsal segment.

Schmaus (8) in the same year published a somewhat similar case, and Pfeiffer (9) in 1891 described descending degeneration in the postero-median as well as in the postero-external columns,

<sup>1</sup> It is well known that in many animals, *i.e.* the rat, mouse, guinea-pig, squirrel, kangaroo, etc., the pyramidal tracts descend in the posterior columns, but animals with such an arrangement are not, of course, considered in this communication.

whilst Barbacci (10) about the same time showed in a case of transverse lesion at the seventh dorsal spinal segment degenerated fibres from that level downwards on either side of the postero-median septum as far as the lower sacral region. Also Bruns (11) in 1893 figured descending degeneration\* in the postero-external columns throughout the first five upper dorsal segments.

Moreover, about 1894, Gombault and Philippe (12) discovered a small triangular superficial tract which degenerates downwards in the posterior columns of the sacral region; and two years later Hoche showed, as a result of compression at the level of the seventh dorsal segment, not merely degeneration in the comma tract as low as the fifth lumbar segment, but also degenerated fibres at the peripheral part of the posterior columns (superficial bundle of Hoche), which could be traced continuously through the oval field of Flechsigs in the lumbar region, and the triangle of Gombault and Philippe in the sacral region as low down as the *conus terminalis*. In another case he found the comma tract degenerated from the seventh cervical to the twelfth dorsal segment, and traces of the superficial bundle from the seventh cervical even as low down as the *filum terminale*.

In the same year (1896), Drs Alexander Bruce and Muir (13) of Edinburgh published a case in which, as a result of a lesion in man in the lower dorsal region, there was definite degeneration along this particular set of fibres, which extended from the twelfth dorsal segment in close proximity to the postero-median septum through the lumbar to the lowest part of the sacral region. To this tract they very happily gave the name of the *fasciculus septo-marginalis*. A little later Dr Bruce (14) showed that this tract, together with the *cornu-commissural tract* (of Pierre Marie) is not degenerated in certain cases of *tabes*. He gave excellent photographs of the course and position of these tracts as stained in such cases by the Weigert-Pal method, reproductions of which are here given for the sake of comparison, and a reference to which will save further description.

Obersteiner (15) has also drawn attention to a tract of fibres occupying an almost identical situation, which he names the *dorso-median sacral bundle*.

Bischoff (16) in 1896, Flatau (17) in 1897, Zappert (18), and Quesnel (19) in 1898, also threw further light on this

subject. A little later (in 1899), Déjerine (20), who had published several accounts of similar descending fibres, published in conjunction with Théohari (21) a masterly summary of our knowledge of these fibres up to that time. They described and figured as a result of compression at various levels in the spinal cord, not only descending degeneration in the comma tract area and immediately adjoining portions of the postero-external columns, but also similarly affected fibres occupying the superficial area described by Hoche in the dorsal region, the oval field of Flechsig in the lumbar region, and the triangular area of Gombault and Philippe in the sacral region. A reference to Fig. ii., Pl. 3, which is a reproduction of some of their diagrams, be of interest.

According to investigations carried out more recently (1902) by Marburg (22), traces of the posterior cervical roots may be followed as low down as the dorso-median sacral bundle; from the cervical region descending fibres can be traced into the upper sacral region along the lateral part of the posterior columns. These fibres undergo a "ventral thickening" at the neck of the posterior horns, and a "dorsal thickening" in the postero-external portions of the postero-external columns. According to Marburg some fibres also pass in continuity from the dorsal thickening to the posterior para-median septum, and thence along the postero-median septum down into the lowest sacral region. In agreement with many others, he describes some of these fibres as arising in the cells of the neck and apex of the posterior horns (therefore endogenous) and ending at various levels lower down in cells in the same regions (also Hoche).

However, van Gehuchten (23), after section of the first and second posterior cervical roots in rabbits, could not trace by the Marchi method degenerated fibres lower down than three spinal segments, whereas after similar section of the eighth cervical and first dorsal posterior roots he could by the same method trace descending degeneration for eight spinal segments. It is interesting to remember that Flatau (17) in the dog, on section of the same roots, obtained the same extent of degeneration, and that Margulies (24) in the monkey obtained on cutting a posterior root, descending degeneration over nine spinal segments, in each case in the comma tract area. Farquhar Buzzard (25) has also published a case of division of the third lumbar pos-

terior root in man which showed degenerated fibres extending below into the lowest sacral region.

It will be seen from the above that two chief sets of fibres show under certain circumstances descending degeneration in the posterior columns of the spinal cord; one set lying in close apposition to the inner margin of the posterior horns and in the comma tract area; the other set lying more mesially in the posterior median columns.

Whether these fibres are to be considered as being entirely of endogenous origin, as is supposed by Daxenberger, Dufour, Margulies, Pierre Marie, Mayer, Philippe, Schäffer, Tooth, Worotynski, Gombault, etc., or whether with Bruns, Flatau, Redlich, Reimers, Schültze, Singer, Zappert, etc., they are to be considered as exclusively arising from the descending branches of the posterior roots, therefore of exogenous origin; or whether, as the writer considers with Dejerine, Müller, Russell, Sottas, Wallenberg, etc., they are both endogenous and exogenous, need not be gone into here.

The discrepancies in the descriptions by the various authors as to the exact level at which the oval field of Flechsig or other tracts appear or attain their maximum development are, as Bruce has pointed out, no doubt due partly to the differences in the nature and situation of the lesion, and partly to the different methods of investigation employed. To this should, I think, be added the fact that all these tracts contain short, intermediate and long fibres; that, in a word, they are really a series of tracts superimposed; and that therefore it is impossible in one section either to divide all the fibres of a given tract or to produce degeneration in the whole length of any fibre.

#### STIMULATION OF THE POSTERIOR COLUMNS AND POSTERIOR ROOTS.

In a research carried out on monkeys and dogs about ten or twelve years ago, the results of which were published in the *Philosophical Transactions of the Royal Society* for 1895, the writer (26) showed that whereas stimulation, say of the third lumbar anterior root, produced rapid extension of the whole hind limb of the same side in the above animals, stimulation of the corresponding posterior root, or still more of the corresponding part of the posterior columns, gave a strong, though

slow reflex contraction of the whole hind limb. The anterior and posterior columns were inexcitable, and it was proved by a process of division and elimination of the various possible reflex paths for the above reflex movements, that the nerve impulse originated by the stimulus applied to the posterior root or the posterior columns passed outwards to the motor roots partly through the same segment, but largely down the posterior columns and out along several segments below the level stimulated; and that the maximum effect even passed out along the second and third segments below the level stimulated. This result, which has been confirmed in the cervical region by Sherrington, and also by others, seems to the writer to throw a strong side light on the presence and function of some of the fibres in the posterior columns which degenerate downwards.

DESCENDING DEGENERATED FIBRES IN THE CERVICAL AND DORSAL  
REGIONS OF THE SPINAL CORD EXTENDING DOWNWARDS FROM  
A LESION IN THE THALAMIC REGION.

About two years ago, at Sir Victor Horsley's suggestion, I did some work for a particular purpose on the thalamic region in several monkeys, etc.; and twelve months later was not a little surprised to find, on examining a cord stained by the Marchi method, some fibres showing degeneration in the cervical and dorsal regions of the spinal cord of a monkey. On examining other specimens of the above animals, I found one in which there was a definite set of coarse fibres, which had evidently degenerated from a bilateral lesion in the upper part of the mid-brain adjoining the thalamus, and were situated on either side of the middle line, forming a wedge-shaped mass of fibres, with the apex anteriorly, and spreading out loosely towards the periphery behind, the bulk of the fibres being situated slightly posterior to the anterior apex of the columns of Goll. A reference to the adjoining untouched photographs of the actual specimens will show, better than any description can give, their exact situation.

It will be seen that in the upper cervical region the degenerated fibres occupy a symmetrical position on either side of the postero-median septum. That in section they consist chiefly of coarse fibres, which occupy a wedge-shaped area, with the apex of the wedge slightly behind the anterior angle of the columns of

Goll, whilst posteriorly the base of the wedge shows scattered fibres, reaching here and there to the periphery of the cord. As the fibres are traced down the cord into the dorsal region they become more scattered, and, as a result of the giving off of collaterals, thinner; whilst they pass for the most part towards the posterior periphery of the cord. At the same time, the angle of the tract opens out so that the fibres, now in section, assume that of a wide angle, or even that of a boomerang.

These fibres had evidently degenerated from at least the upper part of the mesencephalon down to the lower dorsal region. Their path above the decussation of the pyramids will be given in a later communication, in conjunction with some other facts elicited in that research. But the fibres under discussion could be traced in the posterior columns degenerating downwards, and passing into the dorsal region, and in one or two cases traces of similar fibres could be seen actually extending into the lumbar and sacral regions. The specimens, photographs of which are here given, were obtained from the monkey, and a brief note of the fact was sent to the Physiological Society in January last. Since then I have seen, in five or six other cases, fibres similarly degenerated and occupying the same position, though as yet I have not seen them so well marked as represented in the present case, the reason for that being, in all probability, the particular method of operation adopted in the research mentioned above, in which the endeavour was to produce merely a small and limited lesion in the thalamic region or regions. Hence such fibres, if damaged at all, would usually be damaged only in small numbers.

Further work has been carried out with the view of elucidating other points in connection with this particular tract, but it is not yet complete.

The fibres described above must, of course, be long endogenous fibres, which have their origin high up in the mid-brain, or even slightly anterior to this, in the thalamic region, and descend as far as the lower dorsal region, or even lower. The fact that the tract diminishes as it descends in the spinal cord, the fibres becoming smaller and more scattered, occupying a fan-shaped area below the upper dorsal region, would show that the fibres are composed of, as is usually the case, short, intermediate and long fibres; and, as in a somewhat analogous case, that of the posterior longitudinal bundle, it would be



impossible, as Edinger has pointed out, with one section to cause degeneration in all the fibres constituting that tract, or in the complete length of the fibres constituting the tract.

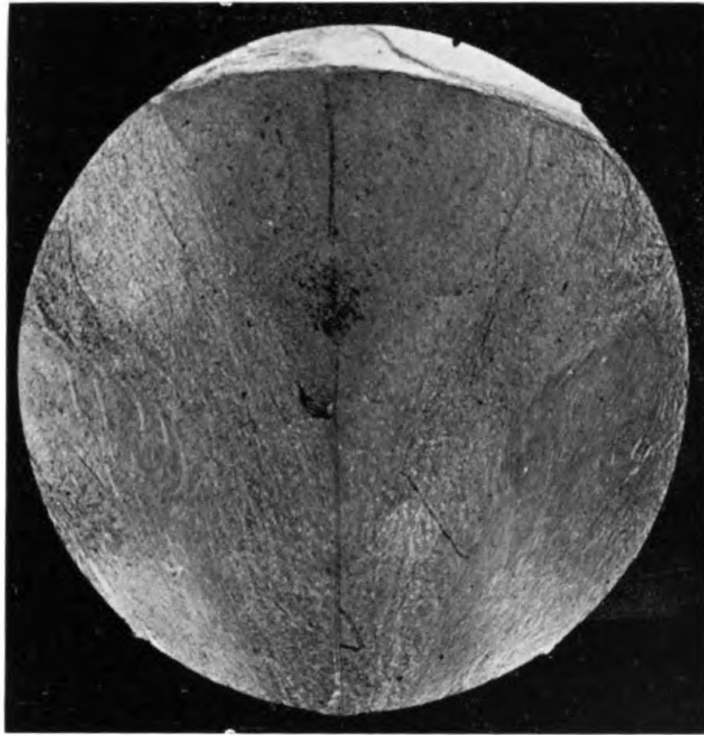
With regard to their function, I feel it necessary to raise a note of warning with regard to the loose employment of the term "descending tracts," or "descending fibres," which is so frequent in text-books and in conversation. It, of course, by no means follows that because a tract undergoes secondary degeneration downwards that therefore it conveys nervous impulse downwards. A good illustration of this is the fact that if a posterior root be cut on the peripheral side of its spinal ganglion, the fibres degenerate towards the periphery (the ordinary Wallerian law), whereas, of course, it is well known that these same fibres must convey nervous impulses in exactly the opposite direction. Therefore, although the fibres described above by various observers, and the set of fibres to which I have called attention in the present paper, degenerate downwards, it by no means follows that they convey impulses downwards. There is a strong presumption that they do convey impulses downwards, but there is no actual proof at present. It may be that they serve to couple up movements of the eyes, or movements which have their centres in the mid-brain, with movements of the hand and lower portions of the body. It may be, also, that they are merely long and short associational fibres subserving in some instances co-ordination, or, of course, their function may be a mixture of the two. Also, van Gehuchten has suggested that they may serve to innervate the lower organic functions. Further work on this point is much needed.

#### CONCLUSIONS.

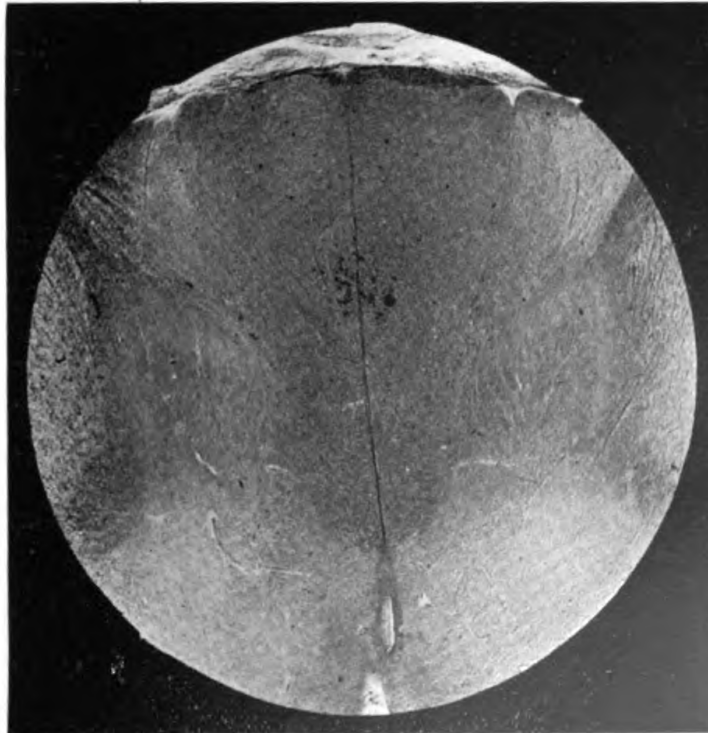
From the above considerations it follows that apart from the bundle of fibres which under certain circumstances undergo secondary descending degeneration in the posterior columns of the spinal cord as described by many observers, viz.: (1) a set of fibres in the postero-external columns occupying the comma tract area and the immediate vicinity of the inner margin of the posterior horns; and (2) fibres lying more mesially in proximity to the postero-median septum—there is also, as shown in the accompanying photographs of the monkey's spinal cord, a well-

DESCENDING DEGENERATIONS IN COLUMNS OF GOLL EXTENDING DOWNWARDS  
FROM THE THALAMIC REGION IN A MONKEY (RHESUS).

PLATE 1.



Level of Cervical iii.



Level of Cervical vi.



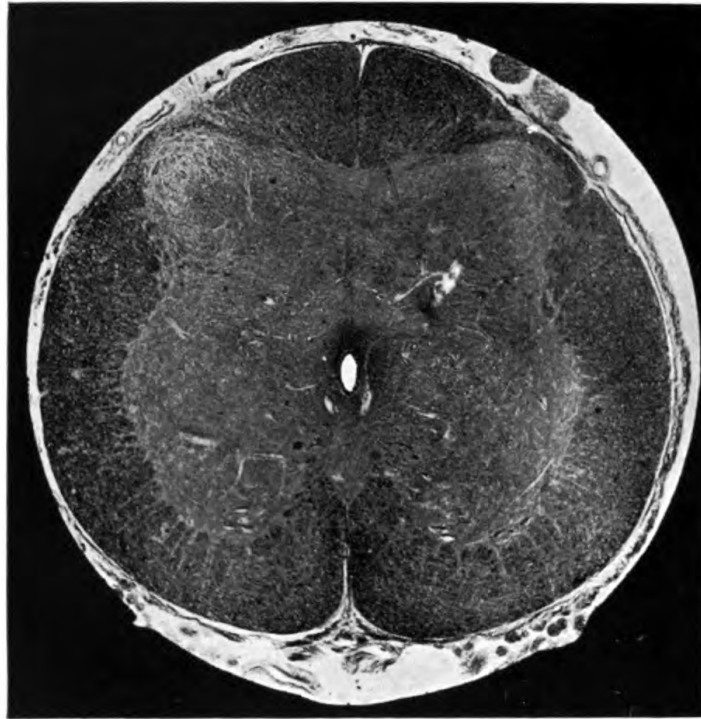


Level of Dorsal ii.

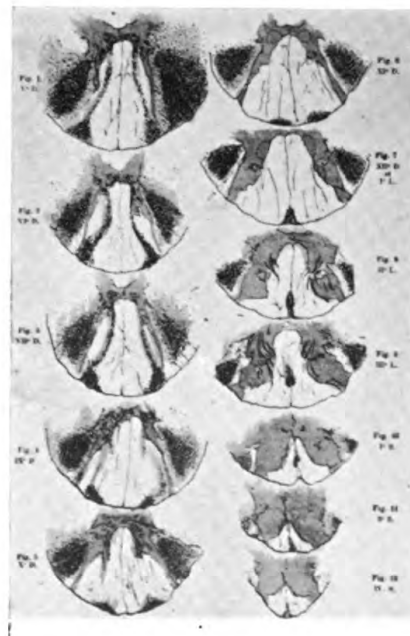


Level of Dorsal viii.





Upper Sacral Region.



After Déjerine and Théohari.



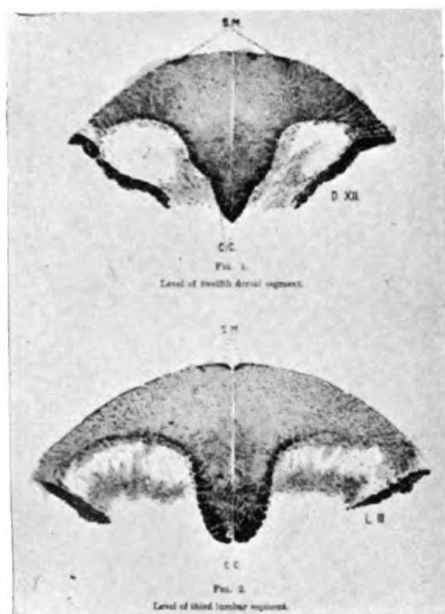


Fig. 1.

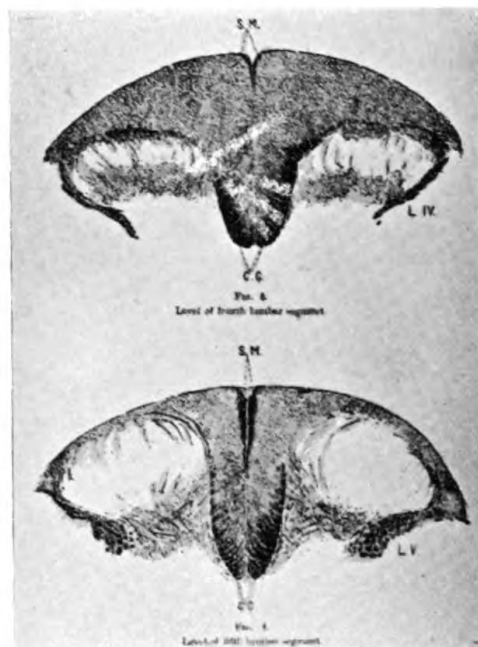


Fig. 2.

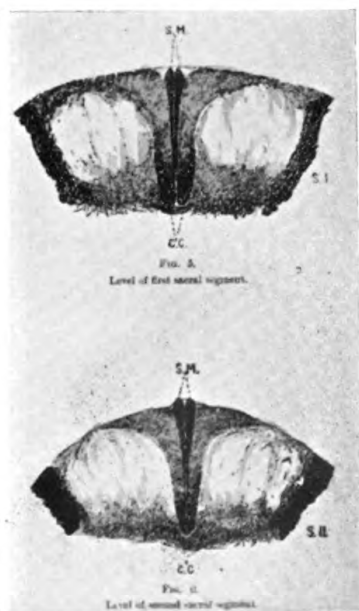


Fig. 3.

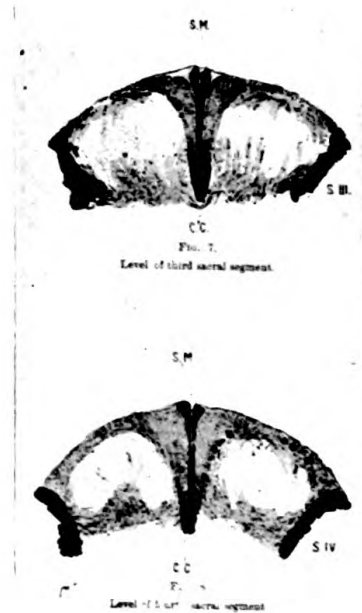


Fig. 4.

After Alexander Bruce.





marked tract, wedge-shaped, in section lying near the middle line in the columns of Goll throughout the cervical region. Above, these fibres originate probably from the thalamus, but in any case as high as the upper (cerebral) end of the mid-brain, whence they can be traced through the brain-stem down into the posterior columns of the cervical region. Below this the fibres become fewer, smaller, and more scattered, and some of them can be traced into the dorsal region, becoming continuous with the superficial bundle described by Hoche, and even occasionally into the oval field described by Flechsig in the lumbar region, and into the triangular bundle described more particularly by Gombault and Philippe in the sacral region.

This tract consists of short, intermediate, and long fibres; but in each area described above there are other scattered fibres which degenerate upwards.

Though the above mentioned fibres undergo secondary degeneration downwards, there is merely at present a strong presumption that they convey nerve impulses downwards, but as yet no proof of this.

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### THE HOMOLOGIES OF THE ROLANDIC REGION.

By ALFRED W. CAMPBELL, M.D., Rainhill Asylum, Liverpool.

Two questions will be considered in this paper. They concern, first, the morphological equivalent of the fissure of Rolando in the lower mammalian brain ; secondly, the sulcus in the Primate brain interchangeable with the lower mammalian sulcus cruciatus. The questions form integrant parts of a widely discussed problem having many correlative and important bearings, that of the homologies of the "regio rolandica." Proof of the stubbornness with which the same problem has opposed resolution hitherto, is afforded by the wealth of controversies it has originated. From a historical narration of the discrepancies in the various propositions submitted on this topic I claim exemption, because, in my belief, the lack of positive result from which we suffer is attributable, in great measure, to disregard of the use and value of the microscope as a discriminating agent ; and in offering conclusions founded mainly, I may almost say entirely, on histological observations, I am to a large extent turning new ground.

First, let us look for the antecedent of the fissure of Rolando in the lower mammalian brain.

In a work communicated to the Royal Society in November, 1903, I emphasised the fact that in both man and the manlike ape, the fissure of Rolando, or in more precise terms, its floor, formed a sharp dividing line between two territories bearing utterly dissimilar types of cortex ; these I designated the pre-central and post-central areas. Into details of the cortical architecture of these areas I need not again enter ; suffice it to say, that the pre-central cortex is characterised by the giant cells of Betz, or ganglionic cells of Dr Bevan Lewis. This fact of histology provides us with a master key to the settlement of the homologue of the fissure of Rolando in animals

lower than primates in the generic scale, and it is one of which I have taken the fullest advantage in this exposition. For, when I made an exhaustive examination, in serial sections, of the nerve cell and nerve fibre architecture of the cortex of *Felis Domesticus* and *Canis Familiaris*, I discovered types resembling, in close particulars, the pre-central and post-central types of the primate brain; I observed, further, that the fields bearing these types were divided, not by the sulcus cruciatus, but by two distinct sulci, one dorsal, shallow and insignificant looking, called the "compensatory ansate," the other lateral, prominent, and well known to us by its name "coronal." Now the proposition I have to submit is that the combination of the "compensatory ansate" and "coronal" sulci of *Carnivoræ* is the forerunner of the fissure of Rolando.

I will first describe the compensatory ansate fissure. Common to many members of the Carnivore family, in *Felis* and *Canis*, it occurs as an isolated fissure, grooving the posterior limb of the sigmoid gyrus, midway between the cruciate and main ansate sulci; although better developed in *Canis* than in *Felis*, from an inspection of a series of brains I am satisfied of its stability in both animals. Then, in a microscopic section made at right angles to the fissure, the manner in which the Betz cells cease the moment the sulcus is reached, is an exact reproduction of what may be seen in a similar section of the Rolandic cortex in Primates.

Next, regarding the sulcus coronalis; in their great work on the histology of the central nervous system, Professor and Madame Vogt do not mention cortical differences between the two gyri bounding the sulcus coronalis. Now, differences do exist; I think that Professor and Madame Vogt have overlooked them, because they examined sections stained for nerve fibres alone. I venture to say that if they will re-examine their specimens, and, at the same time, treat others for the display of nerve cells, they will agree that, in *Felis* and *Canis*, the gyrus sigmoideus and the gyrus coronalis, that is to say, the gyri bounding the sulcus coronalis, are covered respectively by cortex homologous with the pre-central and post-central cortex of primates. In association with this structural affinity, I firmly believe that a corresponding analogy in function exists. Indeed, that the gyrus sigmoideus, like the gyrus precentralis, is endowed with

motor attributes is indisputable; and that the gyrus coronalis, like the gyrus postcentralis, is a sensory end-station and not part of the motor area, as has been supposed, is rendered likely, apart from evidence which I have set forth at length elsewhere, by Tschermak's experiments on the Cat, which show, or seem to show, that this gyrus is a terminus for the cortical lemniscus.

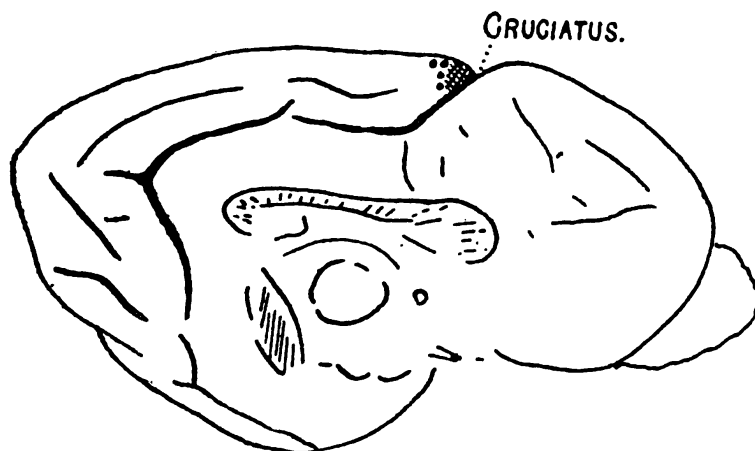
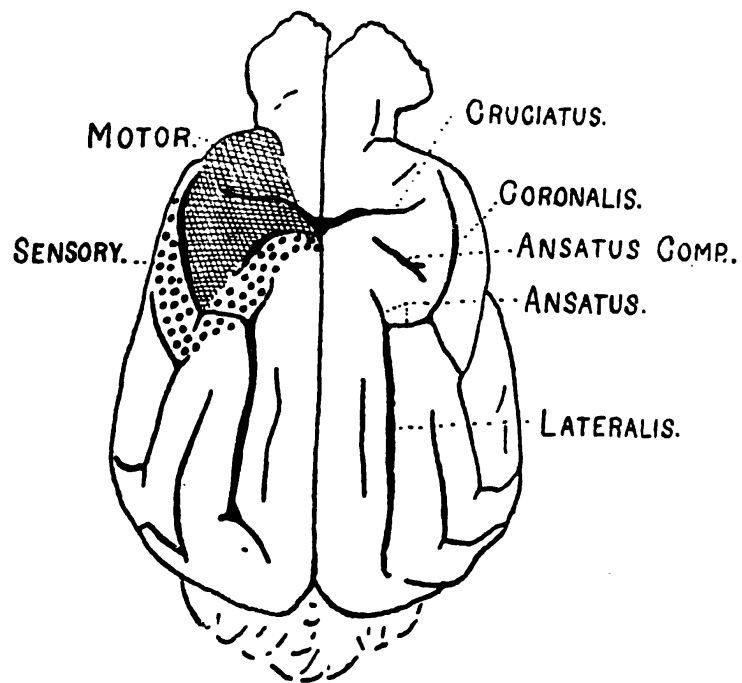
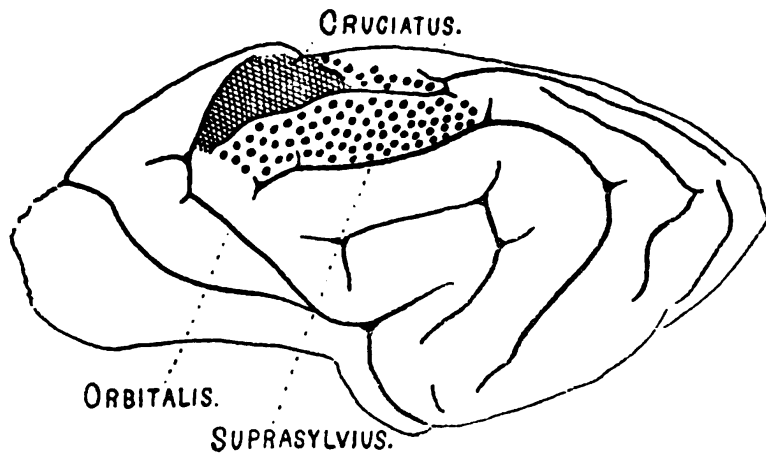
But, it may be asked, how can the fissure of Rolando, which is single, be preceded by two distinct sulci? The explanation is not difficult. We know that for some period during embryonic growth the Rolandic sulcus consists of two portions, an upper and a lower; we know further, that in the fully developed brain, the superior annectant gyrus or buttress is the remnant of the antenatal division, and that occasionally this gyrus remains on the surface, so making the separation permanent. Again, seeing that the buttress guides the experimenter to the point where arm and leg areas meet, we might also say that a physiological subdivision exists. Evidence, therefore, of a reproduction in man of the dual phylogenic condition is not wanting.

Summed up, my proposition is as follows. The cruciate zone of Carnivoræ, becomes the pre-central gyrus of Primates. A field behind the cruciate zone, covering the marginal gyrus partly, but the coronal mainly, develops into the post-central gyrus. The "compensatory ansate" and the coronal sulci are homologues of the upper and lower segments of Rolando respectively, and the intervening substance is moulded into the superior annectant gyrus or buttress.

In the accompanying reproductions of outline drawings of a dog's brain, most of the points mentioned heretofore are illustrated.

Turning to the second portion of our problem, the definition in the primate brain of a sulcus interchangeable with the sulcus cruciatus; obviously from what has just been written, I renounce the common belief that the sulcus cruciatus is the forerunner of the fissure of Rolando, and I will now state further histological reasons in support of my objection.

We have found in our study of the primate cortex, that all the fields we can point to, of known function, are deposited in close relation to stable and important sulci; moreover, these sulci act as territorial limits in, at any rate, one direction; thus,

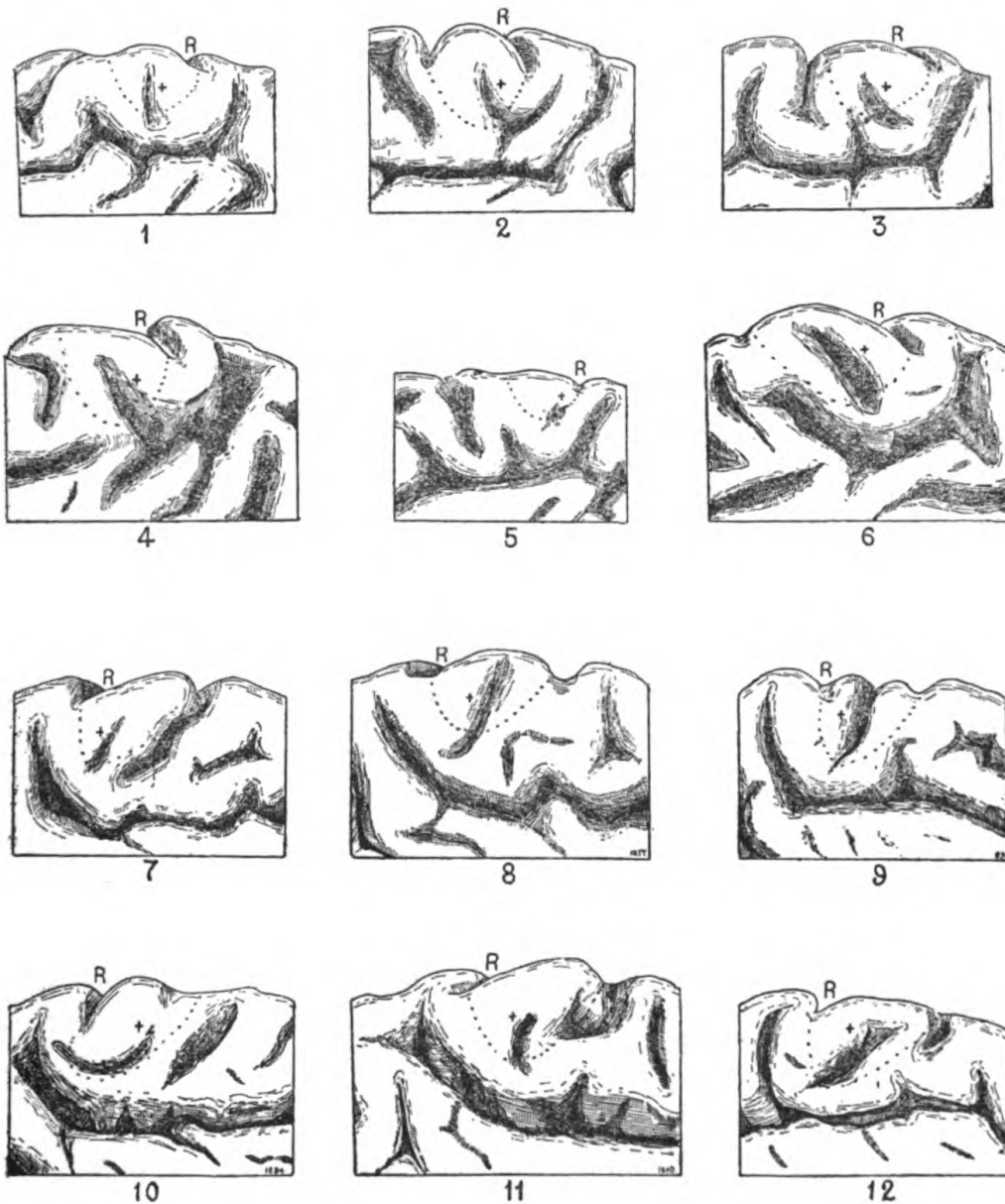


FIGURES OF DOG'S BRAIN REFERRED TO IN TEXT.

the olfactory area is restricted in its spread by the fissure rhinica; the "stem," which we may consider the fundamental component of the calcarine fissure, checks the expansion of visual cortex on to the post-limbic gyrus; the auditory area is closely related to a part of the fissure of Sylvius; and, lastly, strong grounds exist for assuming that the fissure of Rolando acts as a dividing line between motor and sensory cortex. Plainly in the case of all these fissures, two totally different types of cortex cover the apposed walls. Now, in the lower animal, my histological studies point decidedly to a repetition of the association between external morphology and functional distribution; but if, in applying our knowledge to the motor area, we accept the sulcus cruciatus as the homologue of the fissure of Rolando, these anatomical and physiological demands are not satisfied. The sulcus cruciatus has none of the characters of a territorial limit, it is deposited in the midst of the motor area, both its walls are clothed by one type of cortex; in short, it differs fundamentally from the fissure of Rolando.

For the final settlement of the question, we have only to discover and fix the remains of a sulcus cruciatus in the primate, and for my part, I believe a remnant does exist, indeed, considering the prominence of the sulcus in the lower animal, it would be remarkable if it were otherwise. The fissure I have in view is to be sought on the oval or paracentral lobule; it lies immediately below and in front of the upper extremity of the fissure of Rolando; it is so small that it is perhaps better called a fissuret; and it is not to be confused with the anterior boundary of the paracentral lobule, namely, the preoval or paracentral fissure, nor with the inconstant sulcus marginalis. As is the case with almost every fissural element, its appearance is rarely the same in any two brains; in its commonest form, it is an isolated, shallow, vertical or oblique furrow; occasionally it lies horizontally, and not infrequently, instead of being isolated, it runs into the callosomarginal fissure; at times it is triradiate in shape; and, lastly, it may only be represented by a mere dimple. In spite of these morphological variations, however, the truth remains that the fissuret is never altogether absent, at least that is what an inspection of several hundred successive hemispheres suggests. In the Ape's brain, too (Chimpanzee and Orang), I have recognised a corresponding furrow. But while the morphological relations

of this fissure accord with the suggested homology, it is the microscopic structure of the investing cortex which, to my mind, almost places the matter beyond dispute. For, just as in the



lower animal, the deposit of giant cells clings to the sulcus cruciatus, so it is with this fissure. In mapping out the distribution of the motor area I have examined the paracentral lobule, in serial sections, quite a dozen times in man and three



times in the anthropoid ape, and in every case I have detected clusters of giant cells in the walls of the fissure; indeed, so constant is the association, that this furrow, insignificant as it may appear, is always a safe macroscopic fingerpost to the distribution of the giant cells on the mesial surface of the hemisphere.

On previous page is a reproduction of a series of drawings illustrating some variations in the disposition of the "sulcus cruciatus hominis." The six upper drawings were made from right, the six lower from left hemispheres. The field pictured includes the lobulus paracentralis and the posterior portion of the callosomarginal sulcus.

R. indicates the position of the fissure of Rolando, + the sulcus cruciatus, and the dotted line encloses the area occupied by cells of Betz.

### **SOME ASPECTS OF ALCOHOLISM.**

By A. HILL BUCHAN, M.A., M.B., M.R.C.P.E.

I HAVE in the first place to express my best thanks to Dr Alexander Bruce for very kindly allowing me to make the following analysis of the records of cases of alcoholism which were under his charge in the ward for incidental delirium in the Royal Infirmary of Edinburgh. I have also to thank him for valuable advice.

The period embraced is about five years. A complete survey of all the cases or of all aspects of the cases has not been undertaken, but only certain features of uncomplicated delirium tremens and alcoholic insanity. The occurrence of neuritis will be considered separately.

There are special difficulties in the study of such cases, and unfortunately these are often most apparent in those likeliest to be of interest. Complete physical examination, especially of the nervous system, is often during the acute stage of the illness impossible. One has to be content in recording such signs and symptoms as the state of the individual at the time permits. In the case-books there are necessarily many blanks, and

in drawing up statistics one has not a uniform number of entries to go upon for each point discussed.

I. Cases conforming more or less to the delirium tremens<sup>1</sup> type.

To draw a sharp dividing line between D.T. and cases of simpler forms of alcoholic excitement on the one hand, or some cases of alcoholic insanity on the other, I do not believe to be always possible. There are transitional cases in both directions. The term D.T. in literature has also not been fixed in its application. Magnan would restrict it to a very severe type of case associated with pyrexia—a restriction which would reduce our list to small numbers. Other writers would exclude all cases in which actual delusions as apart from sensory hallucinations are present.

There being no single symptom pathognomonic of the disease, one may briefly indicate the general symptom-complex which it is intended the term should indicate in the following pages. It may be described as an event in the history of chronic alcoholism, usually following a special drinking bout, and characterised by prodromal symptoms such as fear, præcordial distress, tremor, disturbances of vision. These symptoms pass more or less rapidly into those of the fully developed attack, which is marked by great mental terror, varied sensory hallucinations, and in some cases delusions—occupation delirium, etc. There is loss of sleep, complete or nearly so, till the occurrence on the second, third, or fourth day or later of a critical sleep, from which the patient wakes in a condition nearly normal as regards his mental faculties, the bodily symptoms being also greatly improved. The crisis may not be so defined and the delusions may tend to recur in the evening which are absent during the day. Relapses may occur or the process pass into some form of alcoholic insanity.

The total number of cases which it appeared might fairly be grouped in this division was 230, 209 of these being men and 21 women.

*Age of the patients :—*

The average age of the men was 39·1 years.

„ „ women, 39·2 „

<sup>1</sup> Delirium tremens will be referred to as D.T.

TABLE I. shows the distribution according to different ages.

	20-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61-65	66-70	71-75	76—Years
Men . .	7	28	44	31	21	26	24	4	3	1	...	1
Women .	1	7	6	7	2	3	2	...	...	2	...	1

*Occupation.*—The occupations were very varied, as seen from Table II.

(a) *Male Cases.*

1. Those specially with spirit trade, etc. :—
  - Workers in Breweries, 4.
  - „ „ Public houses, 9.
  - „ „ Hotels, 2.
  - „ „ Wine stores, 2.
  - Grocers, 3.
  - Butlers, 2.
2. Other occupations :—
  - Bakers, 5.
  - Book-keeper, 1.
  - Cab drivers, 3.
  - Car driver, 1.
  - Cabinetmaker, 1.
  - Clerks, 10.
  - Coachman, 1.
  - Collector, 1.
  - Commercial travellers, 6.
  - Compositor, 1.
  - Cook, 1.
  - Coopers, 3.
  - Cutler, 1.
  - Dairymen, 3.
  - Draughtsman, 1.
  - Draper, 1.
  - Electrician, 1.
  - Engineer, 1.
  - Farmers, 2.
  - Fishmongers, 2.
  - Footman, 1.

2. Other occupations, *continued* :—

- Gardener, 1.
- Glass-blower, 1
- Hammerer, 1.
- Hawker, 1.
- Insurance inspector, 1.
- Joiners, 5.
- Labourers, 5.
- Lorryman, 1.
- Mason, 1.
- Messenger, 1.
- Nurseryman, 1.
- Painter, 1.
- Policeman, 1.
- Riveters, 3.
- Purser, 1.
- Salesmen, 2.
- Stationer, 1.
- Strapper, 1.
- Tailors, 2.
- Teacher, 1.
- Tinsmith, 1.
- Tobacconists, 2.
- Students, 2.
- Upholsterer, 1.
- Warehouseman, 1.
- Wiremaker, 1.

(b) *Female Cases.*

- Actress, 1.
- Domestic servants, 2.
- Housewives, 8.

*Heredity.*—There are notes regarding this in 19 male cases. These are summarised in Table III. In the first column are entered the members of the family addicted to alcohol; in the second those who suffered from insanity; in the third, who had some other nervous affection.

TABLE III.

Case	Family History of Alcoholism.	Family History of Insanity.	Family History of other Affections of Nervous System.
1	Grandmothers (maternal and paternal), grandfather (paternal), brother.		
2	Uncle, brother.		
3		Father, uncle.	Aunt.
4			Mother, sister.
5	Father.		
6	Father, brother.		
7	Uncle.		
8	Father.		
9	Father, mother, two brothers.		
10	Brother.		
11	Father.	Cousin.	
12		Aunt.	
13		Uncle.	
14	Grandfather, father.		
15	Two brothers.		
16		Brother.	
17	Brother.		
18		Sister.	
19		Aunt.	

Thus a family history of alcoholism is more frequent than that of other neurosis.

*The form in which alcohol was taken was as follows :—*

Whisky, . . . . .	in 46 cases.	Whisky and port, . . . . .	in 1 case.
Whisky and beer, . . . . .	25 „	Beer, . . . . .	2 cases.

Whisky and rum, 2 cases. Stout, "no whisky," (?) 1 case.  
 Whisky, rum, and beer, 2 „ Brandy, . . . 1 „  
 Whisky, rum and sherry, 1 case. Methylated spirits, 1 „

Whisky formed part, in all likelihood the chief part of the drink in 93·3 per cent. of the cases in which a note was taken regarding it.

We shall return to the question in dealing with the neuritis cases.

*Quantity of alcohol taken.* One is struck with the large potations which many patients confess to have taken, and it is not likely that they exaggerate their failings.

The notes of daily averages for individual patients are as follows: Whisky, 5 glasses; whisky and 2 gallons of beer; 8 glasses of whisky and 16 glasses of beer; 1 pint whisky; 10 glasses whisky; 3 pints whisky; whisky, beer, and rum, 1½ quarts; 7 to 8 glasses whisky; 5 glasses whisky; 17 glasses beer; 7 glasses whisky, also beer; 2½ pints beer; 1 glass whisky (?) and beer; 5 glasses whisky; 13 glasses whisky; 3 glasses whisky; whisky, 10 glasses, also beer and rum; 8 glasses whisky; 10 glasses whisky; 19 glasses whisky; 3 glasses whisky; 1 pint to 1 quart whisky; 1 pint whisky; 4 glasses whisky.

*Duration of alcoholism previous to attack.*—Under this heading we have to consider, firstly, the total duration of alcoholic habits, and, secondly, the length of the special drinking bout associated with the attack in question.

Table IV. shows the total duration of alcoholic habits arranged in periods.

DURATION.												
	Months.	Years.										
	3-4	4-1	1-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50
Men . .	1	4	15	16	13	11	5	2	...	1	...	...
Women .	1	...	7	3	3	6	3	1	1	1	...	...

If these figures be sufficient for a generalisation, the average duration of alcoholism before an attack is shorter for women than

for men. The average duration for men is 12·6 years, and for women 10·2 years.

Table V. gives the duration of the drinking bout preceding admission to the Wards.

	DURATION IN DAYS.					
	1-5	6-10	11-15	16-20	21-30	31-40
Men . .	6	16	18	...	25	11
Women .	1	8	3	...	1	2

The majority of the patients had, at least, a week's heavy drinking before the attack commenced.

I could find no unequivocal account in the records of an attack of D.T. originating in one short debauch, without previous alcoholism, such as that recorded by Sander (*Arch. f. Phys.*, 1868, Bd. i. 487).

Table VI. gives the age at which the patients began to drink.

	AGES IN YEARS.										
	10-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61-65
Men . . .	7	20	6	14	9	7	4	3	...	1	...
Women . . .	2	2	4	3	3	2	...	1	...	1	...

The early period at which drinking began in the majority of instances is noticeable. In how many cases the amount of alcohol taken was from the beginning excessive, and in how many intemperance developed gradually out of moderate drinking, we have no means of judging.

Comparing Table VI. with Table I., it is seen that while the five years from 16 to 20 represents the period at which the largest number of patients began to drink, the corresponding period in which the majority of actual attacks of D.T. occurred was 31 to 35, or 15 years later; and while the average ages for commencement of drinking was 26·9 for men, and 28·8 for women, the average age of all cases was 39·1 for men and 39·2 for women. But it must be remembered that many of the cases were not first attacks. One cannot tell at what age the amount of drink taken passed beyond the physiological powers of elimi-

nation, etc., of each individual. Consequently, one cannot draw conclusions as to when the average alcoholic is likely first to develop D.T.

*Relation of onset of attack to continuance or cessation of drinking.*—Perhaps nothing as regards the ætiology of D.T. has been the occasion of so much dispute as this, viz., whether or not abstinence after a bout of drinking be a cause or an essential factor in the production of the attack. The records in hand would afford sufficient evidence to disprove the view that it is a necessary condition. The question as to its being sometimes a cause is more difficult to answer.

I have here extracted those references in the case books which appeared to bear at all on this.

Case 1. Steady drinking for 3 weeks: delirious on admission.

2. Patient admitted to general medical ward on forenoon. Excitement did not appear till following night.

3. Chronic alcoholic: had been drinking for 3 weeks and up to admission. When admitted was seeing animals.

4. Said to have been drinking hard till 5 days before admission, when he ceased drinking. On admission delusions present.

5. Quiet on admission: soon became violent. Ascribed his symptoms to "stopping the drink."

6. Had a fortnight's drinking bout, but 8 days' abstinence before admission.

7. Heavy drinking for 2 months. On day of admission (July 2) drank  $2\frac{1}{2}$  pints in 24 hours. Was sensible on 3rd and 4th; on 4th perspiration and tremor noted; on morning of 5th became excited.

8. Had been drinking for a week: hallucinations present on admission.

9. Drinking heavily for a week. On third day after admission complained of "shadows."

10. Drinking "hard for 10 days": delusions present on admission.

11. Drinking heavily for a month till admission, when hallucinations were present.

12. Drinking heavily till night of admission, when delirious.

13. Drinking for a fortnight till admission, when hallucinations were present.

14. Drinking heavily for a week and till admission, when hallucinations were present.

15. Drinking heavily for 6 days and till admission : hallucinations soon appeared.

16. Drinking for a fortnight : had been seeing beasts for 3 or 4 days before admission.

17. Drinking 10 or 12 days : "seeing beasts" for a week.

18. Had "no drink for 2 days," but drinking for 10 days before that. At first, on admission, quiet—then at night talked nonsense.

Some of these extracts (2, 5, 6, 7, 9, and 18) might be instanced in support of the theory that abstinence plays a rôle in the production of D.T., but one must bear in mind that though it may be noted that no symptoms of mental disturbance were present on admission, in almost all instances when the patient was sent to the ward for incidental delirium, something was observed outside which made it appear likely that the case was one not suitable for a general medical ward.

Cases 1, 3, 8, 10, 11, 12, 13, 14, 15, 16, and 17 show these disorders arising without a period of abstinence.

Patients may naturally like to emphasise the virtues of any abstinence. A day may be fixed on by the friends as the date of commencement of the disease ; but this only means that then symptoms became so obvious as to attract attention ; it does not follow that all was normal before that. The earlier symptoms are but aggravations of those of chronic alcoholism, and may not at first strike the casual observer. The initial hallucinations may be noticeable only during a part of the 24 hours, and the patient give no indication of their presence unless specially watched and questioned.

It seems more likely that in the records of prisons, poor-houses, inebriate homes, etc., data for the adequate discussion of this question will be found.

*Epileptiform Convulsions* have been looked on by some writers as a cause of D.T.

Of a total of 19 cases in which convulsions occurred during the attack, or in connection with the drinking bout preceding the attack, 17 were men and 2 were women. The average age was 35·6 years. 16 recovered, 3 died—a high death-rate compared with the general mortality of all cases. The total duration



of alcoholic habits was: 20 years, 3 cases; 15, 12, and 10 years, each 1 case; 16 months, 1 case; 2 weeks (?), 1 case. The fits generally occurred in the prodromal or earlier stage of the disease. As regards their relation to cessation of drinking, if we take the date of admission to hospital as a fixed point (it being certain that no alcohol was obtained thereafter), we find that before admission 12, after admission 2, and both before and after admission 3 patients had fits. The withdrawal of alcohol is apparently not prejudicial in this respect. It is not unlikely that the number of patients who had fits before admission is greater than the records show, while any seizure occurring after the patient was in the ward would certainly be noted. The fits occurred approximately within 12 hours before admission in 3 cases, within 24 hours in 2 cases, within 3 days in 1 case, 4 days in 1 case, and 5 days in 1 case. The last fit after admission occurred on day of admission in 6 cases, on first day after admission in 1 case, on second day in 1 case, and on third day in 1 case. The total number of fits recorded during the illness: 9 in 2 cases, 7 in 1 case, 4 in 1 case, 2 in 5 cases, and 1 in 5 cases.

As to the nature of the seizures themselves, they varied from epileptiform convulsions of a severe type to slight attacks with but a brief loss of consciousness. One case (female) had 9 convulsions within half-an-hour, and *status epilepticus* was established. D.T. symptoms supervened, but the patient eventually recovered, and was able to leave hospital in 10 days. Magnan has said that when the fits are separated by several hours' interval, they usually pass off without accident, but when they follow one another every 5 or 10 minutes, the patient may succumb at the fourth attack.

In many of the cases it was noted that the patients bit their tongue: in one a distinct aura occurred.

In some instances the delirium immediately follows the fit, while in others the two events are separated by an interval, during which no symptoms suggestive of D.T. may be noticed. Thus, in the case just quoted, the restlessness, incoherence, loss of orientation, hallucinations, etc., developed on the day following the fit. Another patient had 4 fits on 26th inst., on 27th appeared well, on 28th had one fit, and became wild and unmanageable.

In all these cases, with the exception of 2, a temperature

above normal was recorded at some period of the illness, but only a slight degree of fever was as a rule present ( $99^{\circ}$  to  $100.5$ ). In one case the temperature rose to  $104.2$ , in another to  $103$ .

It is difficult to see what proof can be brought forward that convulsions may cause an attack of D.T. in an alcoholic. It is admitted that a chronic alcoholic may have such seizures and D.T. not develop; and when D.T. does appear, it is frequently only after an interval, during which mental symptoms are in abeyance.

*Pyrexia.*—When we arrange the cases according to the highest temperature recorded, we find that the higher the temperature selected, the more does a summer maximum predominate.

TABLE VII. HIGHEST TEMPERATURES.

Cases whose highest temperature was :—

	Jan.	Feb.	Mar.	Apl.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
98—99	9	2	6	6	8	8	14	10	6	12	11	9
99.1—100	4	6	5	13	5	6	2	7	8	10	7	8
100.1—101	2	1	1	2	1	...	3	1	1	1	2	...
101.1—102	1	1	1	3	1	...	1	1	...	...	...	1
102.1—103	...	...	...	...	...	...	1	1	2	...	...	...
103.1—104	...	...	...	2	...	1	...	1	...	...	...	...
104.1—105	...	...	...	1	1	...	1	2	...	1	...	...
105.1—106	...	1	...	...	...	1	...	...	1	1	1	...
106.1—107	...	...	...	...	...	...	3	...	...	...	...	...
107.1—108	...	1	...	...	...	...	...	...	...	...	...	...

From the above it would appear that even a moderate degree of pyrexia is not the rule in D.T. One must bear in mind, however, that these temperatures were taken in the axilla, not in the rectum, as in cases recorded by some observers. The fact that the patients are often bathed in perspiration and are constantly moving about, renders the axillary method not so reliable. This may help to explain the conflicting views expressed in literature regarding the presence of pyrexia. While Magnan limits the term true D.T. to a very severe type of case associated with fever, others have maintained that D.T. is never by itself a febrile process.

The records point to the conclusion that while D.T. may be

accompanied by fever, it is not necessarily so, febrile reaction being generally slight. In preparing this table I have endeavoured to exclude all cases having any complication that might cause pyrexia ; but here again the difficulty of satisfactory physical examination is so great that apart from post-mortem confirmation one hesitates to dogmatise. Clinically, however, there is some evidence which favours the recognition of a special pyrexial form of D.T. In the more characteristic cases the temperature from being normal or nearly so suddenly mounts up to a high point, the pulse becoming correspondingly rapid. There are five cases in the records in which this sudden pyrexia is a prominent feature.

The patient usually succumbs rapidly. In one case, however, which at first appeared to be of this type, the patient recovered. His temperature on admission was normal in the morning, 98·8 in the evening ; next day 99 in the morning, 103 in the evening. On the following morning it shot up to 107·5, the pulse being 140-150. Under ice it was brought down, and after 10 to 12 days of moderate irregular pyrexia the patient got well. Such a result is exceptional.

There were 15 cases of D.T. in which the temperature exceeded 104. The average age was 37·6 years. Three had a history of drinking for 10 years, 2 "for long," 1 for 3 years. Two had abdominal distension. In 3 there is a special note of marked perspiration. Very coarse tremor was noted in 3.

*Pulse Rate.*—The average highest pulse reading in cases which recovered was 95 among the men, and 96 for women. The average in fatal cases was 115 (the first reading on patients' admission is excluded).

The average lowest pulse rate in cases which recovered was 72 for the men, and 71 for women ; in cases which died, 95.

*Urinary System.*—Albumen was noted as present in the urine in 22 cases, *i.e.* in 9·5 per cent. 16 of these were men, and 6 women. One has seen a large quantity of albumen disappear in a day or two ; but the difficulty of obtaining specimens during the acute stages doubtless renders this percentage too low.

*Alimentary System.*—One is struck with the comparative rarity of acute symptoms of gastro-intestinal disturbance, at least of such severity as to cause manifest distress to the patient.

The patients almost invariably are able to retain food and medicine. One would expect the reverse. I do not recollect having seen a patient who could not retain bromide and chloral mixture.

TABLE VIII. summarises the Notes as regards Alimentary System.

	Nausea.	Pain.	Vomiting.	Diarrhoea.	Abdominal Distension.	Icterus.	Results.
Men. }	4	9	10	5	4	3	Recovery.
		1	2		3	2	Death.
Women. }		2		1			Recovery.
		1		1			Death.

That among such a large number of acute alcoholic cases jaundice, for example, should have been recorded only 5 times is remarkable.

*Integumentary system.*—Perspiration was specially noted in 22 cases.

*Mental symptoms.*—Hallucinations may be taken as always occurring. There are special notes regarding their presence in 109 cases. In 57 of these a more or less definitely delusional element was also present. In 53 male and 17 female cases the hallucinations were visual; in 23 of the former, however, and 5 of the latter the auditory sphere was implicated as well as the visual. In 10 male and 5 female cases auditory hallucinations alone were noted.

Hallucinations of animals were recorded in 49 male and 10 female cases, only two of these being fatal. In view of a tradition that the smaller the size of the animals seen the worse is the prognosis for the patient, an analysis was made of all the cases of alcoholism during the five years in question. The result is given in Table IX., which, it will be seen, does not bear out this idea.

TABLE IX.

Hallucinations Present.	Recovered.	RESULT.	
		Sent to Asylum.	Died.
Dogs	20	2	...
Rats	15	2	...
Cats	12	2	3
Insects	11	2	...
Horses	8	...	2
Snakes	8	...	...
Mice	6	...	1
Fish	5	...	...
Specks	5	...	...
Lions	4	...	...
Bears	4	...	...
Pigs	3	...	...
Reptiles	3	...	...
Birds	2	...	...
Deer	2	...	...
Kittens	2	...	...
Cattle	1	1	...
Rabbits	1	1	...
Crocodiles	1	...	...
Dragons	1	...	...
" Half-cats, half-birds "	1	...	...
Hedgehogs	1	...	...
Leopards	1	...	...
Lizards	1	...	...
Lobsters	1	...	...
Monkeys	1	...	...
Snails	1	...	...
Tigers	1	...	...
Weasles	1	...	...

In no case of simple D.T. were hallucinations connected with the abdominal viscera referred to. One patient who had such did not present mental symptoms typical of D.T. He had eventually to be sent to an asylum.

Occupation delirium was noted in 22 male cases which recovered. Delirium of a more markedly systematised character

was recorded in 9 non-fatal male cases; while in 3 cases, 1 of which was fatal, something of the nature of an explanatory delirium was observed.

Suicidal tendencies were present in 14 male cases, 2 of which died; but it is often difficult in this disease to tell whether an act is due to true suicidal impulse or is a mere accident arising from the terrifying nature of the hallucinations.

Excitement was specially marked in 69 male cases, 6 of which died, and in 10 female, 2 of which died.

Ideas of grandeur were not recorded in any case of uncomplicated D.T.

In 23 cases it is mentioned that mental symptoms persisted after the occurrence of sleep, 3 of these cases being fatal.

*Mortality.*—The prognosis of moderately severe D.T. is usually good. 16 cases proved fatal out of a total of 230, or 6·97 per cent.

The average age of fatal cases was 40·5 years, as compared with 36·9, the average age of non-fatal cases.

The larger number of deaths occurred in the warmer months.

TABLE X. FATAL CASES.

Number of Fatal Cases.	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
4												
3					—	—			—			
2		—			· ·	· ·	—	—	· ·			
1	· ·	· ·			· ·	· ·	· ·	· ·	· ·		· ·	
0	—	· ·			· ·	· ·	· ·	· ·	· ·		· ·	

One had drunk for 13 months; another for 2 years.

The duration of the drinking bout immediately preceding the attack was :—

No. of cases, 2 = duration of bout, 6 weeks.

Do. 1 = do. 4 do.

Do. 1 = do. 12 days.

Do. 1 = do. 1 week.

*Time of Death after admission to hospital.*

1 case . . . . .	13 days.
5 cases . . . . .	2 „
1 case . . . . .	10 „
1 „ . . . . .	12 „
2 cases . . . . .	6 „
5 „ . . . . .	within 24 hours.

One had hepatic cirrhosis with jaundice; in one vomiting and pain were marked symptoms; two had abdominal distension, in one of them diarrhoea with pea-soupy stools was present.

TABLE XI. HIGHEST TEMPERATURE IN FATAL CASES.

	Jan.	Feb.	Mar.	Aprl.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Up to 99	..	...	...	...	2	...	...	...	...	...	...	...
99·1—100	...	1	...	...	...	...	1	1	...	...	...	...
100·1—102	...	...	...	...	...	...	...	...	...	...	...	...
102·1—103	...	...	...	...	...	1	1	...	2	...	...	...
103·1—104	...	...	...	...	...	1	...	...	...	...	...	...
104·1—105	...	...	...	...	...	...	...	1	1	...	...	...
105·1—106	...	1	...	...	...	1	...	...	...	...	1	...
106·1—	...	...	...	...	...	...	1	...	...	...	...	...

## HIGHEST PULSE RATE.

140 and below	. . .	2 Cases
130 „	. . .	1 „
120 „	. . .	9 „
110 „	. . .	1 „
100 „	. . .	1 „
90 „	. . .	1 „
80 „	. . .	1 „
70 „	. . .	0 „

In 4 cases albumen was present; a high proportion compared with that of non-fatal cases.

Five had hallucinations; 2 hallucinations and delusions. In 2 it is noted that the hallucinations affected both visual and auditory spheres. One had visions of animals; 3 had ideas of persecution; 2 had systematised delirium; 1 suicidal tendency; in 3 delirium persisted after sleep. Where orientation was noted, in 2 it was defective as to time and place, in 1 as to persons. In 5 there is special note as to tremor. In those cases in which the condition of the pupils was noted they were dilated.

## Abstracts

### ANATOMY.

#### THE MINUTE STRUCTURE OF THE NERVE CELLS IN MAN.

- (1) (*L'intima struttura delle cellule nervose umane.*) ROSSI,  
*Névrose*, Vol. vi., f. 3, p. 329.

THE author uses the following method:—Pieces of fresh tissue of 3-4 mm. in thickness are placed in a solution of 2 per cent. nitrate of platinum for 24-48 hours; then in gold chloride, 50 per cent.; and after slight washing in distilled water, they are placed in 1 per cent. formic acid for 24 hours in the dark. After a rapid wash in distilled water they are embedded in paraffin. In the nerve cells of the spinal cord the superficial fibres are larger and less numerous. Some fibrils pass from one process into another, but the majority form a network in the cell by anastomoses. Around the nucleus the reticulum is often dense, while towards the periphery the meshes are larger and the fibrils finer. Fibrils can be seen passing through the cell body into the axis-cylinder. In some cells they are thicker in the centre of the processes.

In Purkinje's cells there is a network the meshes of which are much elongated in the protoplasmic processes. The basket-work arrangement of fibres around these cells is stained and does not form any connection with the cytoplasmic reticulum of the cell. In the cortical cells there are large and fine fibrils as in the spinal cord. The large ones of the apical process are directed towards the axis-cylinder and give origin to finer or secondary fibrils, both varieties combining to form a meshwork.

In many cells the neuro-fibrils diverge at the nucleus and surround it by a dense reticulum. Some of the large fibrils pass into the axis-cylinder, giving off finer fibrils and anastomosing with neighbouring ones. The reticulum is usually very evident at the base of the pyramidal cells, and from it the fibrils of the axis-cylinder take origin. In the spinal ganglia the fibrils are fine and form a reticulum the meshes of which become smaller from the cell periphery to the centre. These fibrils are of uniform thickness.

The organised achromatic substance of the nerve cell is formed by the reticulum.

DAVID ORR.



**A CONTRIBUTION TO THE STUDY OF THE FINE HISTOLOGY**

(2) **OF THE NERVE CELL.** (*Contribution à l'étude de l'histologie fine de la cellule nerveuse.*) MICHOTTE, *Névrose*, Vol. vi., f. 3, p. 279.

THE author has examined the fibrils in the nerve cells of the posterior root ganglia, spinal cord, medulla, cerebellum and cerebrum, and gives his results in great detail.

In all nerve cells there is a network which sometimes gives to the cell a reticular appearance; at other times, when there are fewer trabeculæ, a more fibrillar structure. Between these two extremes there are intermediate forms.

A pure reticulum is found especially in sensory cells, *e.g.* retina, root ganglia, olfactory bulb. In the multipolar cells the structure is reticulo-fibrillar.

The fibrils in the cell-body are never independent except in badly stained material. In certain cells which show a network only, all the fibrils of the prolongations enter into communication with the network in which they are lost.

The structural part of the cell protoplasm forms a network of fibrils separated from each other in the cell body, and thus exposing to view the trabeculæ, while in the prolongations where they are pressed together these trabeculæ are hidden.

In the less highly differentiated cells such as the globose type, the fibrils form a network only, but other types are specially modified and adapted for the function of conduction, and in these the fibrils assume greater importance as conducting structures and the trabeculæ are less numerous.

There are no extra-cellular anastomoses and each neuron is independent.

DAVID ORR.

**THE STRUCTURE OF THE MYELIN SHEATH.** (*Sulla struttura*

(3) *della guaina mielinica.*) REBIZZI, *Rev. di Patolog. nerv. e ment.*, Vol. ix., f. 9, 1904.

THE author's method is as follows:—Fixation in formalin, 20 per cent., for at least 24 hours; the nerves are then passed, without drying or washing, into a di-ammoniacal solution of silver nitrate, in which they remain for 24 hours, and are then passed into a solution of formalin, 10 per cent., rendered strongly alkaline.

Washing, dehydrating, and embedding (in paraffin) are performed rapidly. The sections are treated with chloride of gold,  $\frac{1}{2}$  per cent., then passed into hypsulphite of soda, 5 per cent., then washed shortly in slightly acidulated water.

In nerves a reticulum and cone-shaped structures are stained, the apex of the latter lying against the axis-cylinder. The position

of the so-called Schmidt-Lautermann incisures remains unstained, indicating the division between the cylinder cones.

The basal part of the cones is connected with a reticulum at the periphery of each segment. By some methods this is not so well coloured as the apex.

The myelin sheath is apparently of reticular structure, and the walls of the alveoli contain a small amount of protoplasmic substance, which is more abundant at the apices of the segments. This protoplasmic substance may be coagulated by fixation, and dissociated in the embedding process.

The author accepts the view that the Weigert-Pal method colours the small amount of protoplasm present in the walls of the alveoli and in the cones.

In the alveolar walls the myelin consists of cholesterin—which is coloured by the author's method—lecithin and protargon, which remain unstained. Of these two, one probably exists in the wall of the alveoli, while the other is inside them.

With regard to the question as to whether it is the death of the elements or reagents which determine the disposition of the components of myelin, the author thinks it probable that the structure is pre-existent, and fixation and embedding only exaggerate the specific electivity of each element for various reagents.

According to the suggestion of the author, the myelin sheath is alveolar in its peripheral part, and homogeneous in the centre, and the incisures of Schmidt-Lautermann are closed at both extremities.

In the myelin near Ranvier's nodes, probably more cholesterin exists. The fine fibres colour more intensely than the coarser ones.

DAVID ORR.

**CRANIO-CEREBRAL TOPOGRAPHY.** RICHARD J. A. BERRY  
(4) and HUBERT D. SHEPHERD, *Brit. Med. Journ.*, Nov. 19, 1904,  
p. 1382.

IN a paper dealing with the important subject of cranio-cerebral topography read before the Anatomical Section of the British Medical Association and subsequently published in the *British Medical Journal* on November 19th, 1904, we pointed out that the improved methods of observation now at the disposal of the anatomist consequent on the introduction of formalin, the numerous and conflicting statements universally made as to the best methods of determining upon the surface the positions of the great fissures of the human cerebrum, and the great clinical and scientific importance of the subject all alike seemed to demand its reinvestigation.

Our first series of cases comprised an examination of methods associated with the names of, first, Hare and Thane (published in "Quain's Anatomy"); second, Chiene (published in "Cunningham's Textbook of Anatomy"); and third, Reid (published in "Treves' Surgical Anatomy"). In every instance the method of procedure was the same. The brain was thoroughly hardened by formalin injections on the undissected subject; the guiding lines employed by the particular method under investigation were drawn upon the skull and all intermediate portions of bone removed. The meninges of the brain were then dissected off, with the obvious result that the precise relations of the fissures to the guiding lines could be accurately noted. It need hardly be said that, though this method was excessively laborious, it gave the utmost precision obtainable, and constituted a particularly severe proof of the accuracy or otherwise of the particular method under investigation.

Working with Hare and Thane's method, we examined ten cases with the following results. The superior Rolandic point was correctly indicated in 60 per cent. of cases, and in the remaining 40 per cent. the point was indicated as being too far forwards.

The fissure of Rolando, as indicated by the employment of Hare's angle, was crossed in some portion of its extent by the guiding line in every instance.

The Sylvian point was correctly indicated in 70 per cent., but the Sylvian fissure was incorrectly indicated in every instance, the error in each case being the same, namely, that of indicating the fissure as being more oblique than it really is.

The parallel fissure and the parieto-occipital fissure were fairly accurately indicated by this method.

Working with Chiene's method we examined seven cases. This method is a proportional method and professes to constitute a surface guide to the anatomy of the brain. It does not specially indicate the fissure of Rolando, but rather attempts to locate the Rolandic motor area which by this method, contrary to Sherrington's conclusions, comprises the ascending frontal and ascending parietal convolutions. Even with this variation from Sherrington, we only found the motor area correctly indicated in 43 per cent. of cases; it was altogether incorrect in 43 per cent. and only partially correct in the remaining 14 per cent. The Sylvian point was correctly indicated in 71 per cent. and the fissure of Sylvius in 57 per cent. In the remaining cases the error regarding the fissure of Sylvius was the same as in Hare and Thane's method—indicated too obliquely.

Working with Reid's method we examined three cases, but the general results obtained were so incorrect that we did not further pursue this line of research.

A comparison of our results with the three methods investigated shows conclusively that no one of the three methods is in itself sufficiently accurate as to justify its retention to the exclusion of the others. Hare and Thane's method gave us the most uniformly good results, and we therefore recommended the retention of their method as regards the superior Rolandic point and the fissure of Rolando. All the methods were seriously at fault as regards the fissure of Sylvius, and we therefore suggested at Oxford a new method for locating the fissure. As our experience had been that the fissure of Sylvius was always too obliquely indicated, we advised that a line should be drawn from the fronto-nasal suture to the lambda, and divided into thirds, the middle third of this line should correspond to the posterior horizontal limb of the fissure, whilst the junction of the anterior and middle thirds should give the Sylvian point. This method had the obvious advantages of abolishing the employment of millimetres as in Hare and Thane's method, of being proportional and therefore adaptable to any shape and size of skull, and above all, of avoiding the error of too great obliquity found in all the other methods.

Since the publication of the paper, of which this is merely a condensation, we have commenced the investigation of a second series of cases in which our theories, as regards the Sylvian point and fissure, have been subjected to the same searching tests as the methods mentioned. We have also taken the opportunity of re-examining Hare and Thane's and Chiene's methods, but we have found no reason to alter our opinion; we have also examined Anderson and Makin's method and Cathcart's method for the fissure of Rolando. It is yet too soon to speak of the results obtained by this second series of cases, but so far as we have gone, we feel confident that our conclusions are justified, and that our own method of locating the Sylvian point and fissure will be found to be the most reliable.

In a future communication we hope to deal with both series of cases, together with demonstrable proof of the conclusions to be drawn from the same.

AUTHORS' ABSTRACT.

## PHYSIOLOGY.

### THE PROGRESSIVE EVOLUTION OF THE VISUAL CORTEX IN

(5) **MAMMALIA.** F. W. MOTT, *Lancet*, Dec. 3, 1904, p. 1555.

MOTT begins by showing that the more an animal depends for self-preservation on the acuteness of its vision, the greater becomes the complexity of the cell lamination in its visual cortex, this

being especially shown in the increasing development of the pyramidal cell layer, which is found just above the granule cells of the cortex. In the insectivora the visual cortex consists almost only of small stellate or quadrilateral cells with a thin layer of polymorph cells subjacent, amongst which lie a very few pyramidal cells. The rodentia possess a wide panoramic field of vision, and therefore have a somewhat more developed cortex, large stellate and pyramidal cells forming a thin layer above the granule cells. Amongst the marsupials the herbivorous forms have a poorly developed pyramidal layer; while the more acute sighted carnivores have this layer well marked. The ungulates have only an ill-developed pyramidal zone. The carnivores fall into two groups: the canines, with widely set eyes, have panoramic and partly binocular vision—these possess a fairly deep layer of large and small pyramids; the felidæ, having definite binocular stereoscopic vision, possess a further developed pyramidal zone with numerous solitary cells of Meynert. In the primates the cortex becomes divisible into two distinct regions: (*a*) visuo-sensory; (*b*) visuo-psychic, or associational; this region contains many large pyramidal cells, and is most fully developed in man, in whom its maximum development is only attained after birth.

The progressive development of the visual cortex shows a parallel between the extent of the pyramidal cell layer and the evolution of binocular vision.

The striate area, the primary seat of vision in which the optic radiations terminate, does not occupy the entire occipital lobe in the anthropoid apes and in man, but is limited to the mesial surface, especially to the part immediately surrounding the calcarine fissure. The great development of the parietal lobe in man causes an enfolding of the occipital lobe by a phylogenetically new cortex, which is distinguished from the old by the absence of the line of Gennari, the fusion of the two granule cell layers, and the presence of a third layer of pyramidal cells. Elliot Smith has shown that in Fellaheen and Soudanese brains the striate cortex may be present on the external surface of the occipital lobe, closely resembling the condition met with in the gorilla; Mott has found the same anatomical arrangement in the brains of lunatics, as also in a Chinese, a Goanese, a Congo negro, and an Egyptian; so Smith is probably correct in regarding the Affenpalte as not entirely restricted to the brain of the ape. With regard to the terminal distribution of the optic radiation, Monakow states that macular impressions are conveyed to the whole of both occipital lobes, and also even to the angular gyri. Henschen regards the calcarine area as forming a true cortical retina, the lower half of this region corresponding to the lower quadrants of homonymous halves of the retinæ, and the upper half

to similar upper quadrants ; he instances cases in which disease of the lower calcarine region has been associated with hemianopsy of the lower quadrants and *vice versa*. Mott does not regard macular cortical localisation as definitely settled, but thinks that the great development of macular vision in the primates may well be associated with the increase of non-striate cortex, thus favouring Monakow's view. Voluntary eye movements are not lost in cases of bilateral hemianopsy owing to the presence of a definite centre for these in the frontal lobe ; this lies in the ape's brain just in front of the precentral sulcus, separated by an inexcitable area from the head and neck centres. Mott points out that there is much evidence in favour of the view that the frontal lobe in man has to do with the balancing of the body, ataxy is a constant symptom of frontal lobe tumours, and so suggests that this inexcitable frontal zone may have to do with the fusion of visual perceptions, sub-conscious equipoisal sensations and tactile kinæsthetic impressions for the purpose of equilibrating the body. In relation to hemianopsy, Mott further points out the frequency with which the loss of memory pictures and of orientation is associated with this condition ; in all cases in which these co-exist there has been found to be bilateral damage to the occipital lobes, while in those cases in which loss of orientation has been associated with unilateral hemianopsy, there were bilateral lesions of the occipital lobes, but the striate cortex was found intact on one side.

E. H. FRASER.

#### **HISTOLOGICAL STUDIES ON THE LOCALISATION OF CERE-**

- (6) **BRAL FUNCTION.** ALFRED W. CAMPBELL, *Proc. of Roy. Soc.*, Vol. 72, and *Journ. Ment. Sc.*, Oct. 1904, p. 651.

A RESEARCH, based on an exhaustive examination of the cortex cerebri of Homo, some anthropoidea and certain lower mammals, and aiming at the furtherment of the correlation between physiological function and histological structure, is summarised.

With the assistance of the Royal Society, the work, *in extenso* and in book form, will appear in due course.

#### **ON THE EARLY HISTORY OF CEREBRAL LOCALISATION.**

- (7) DONLEY, *Am. Journ. Med. Sci.*, Oct. 1904, p. 711.

To the unwarrantable belief in the existence of "animal spirits" which dominated medical thought from the time of Hippocrates till the beginning of the nineteenth century, the writer attributes

the slowness of the progress that has been made in elucidating the functions of the central nervous system. This animal spirit or ethereal principle which was supposed to pervade the nervous system passed under various names at different times, such as *pneuma*, innate heat, sensitive soul, and even the *vis medicatrix naturæ* of Cullen. One argument for its existence was that the brain, from its appearance, structure, and rich blood-supply, came to be regarded as a secreting gland, and therefore was supposed to secrete the active animal spirit.

Erasistratus, Herophilus, Galen, all distinguished sensory from motor nerves. As early as the second century A.D., Aretæus recognised that injuries on one side of the head produce paralysis of the opposite side of the body, while disease affecting the spinal cord causes paralysis on the same side of the body; but not until the beginning of the nineteenth century did Gall and Spurzheim, the founders of phrenology, first demonstrate the decussation of nerve fibres in the pyramids.

As to the mental activities, Plato recognised three faculties, situated respectively in the liver, heart and brain, by which their appropriate spirits were secreted, while his teaching was adopted by Galen, Vesalius, etc. In the Middle Ages, however, the Arabian physicians placed the different mental faculties in the several ventricles of the brain, this theory being adopted by Duns Scotus and Thomas of Aquin, and referred to by Burton in his "Anatomy of Melancholy."

In the end of the eighteenth century, when chemistry had been advancing with rapid strides, we find that Prochraska, writing in 1784, discredits the theory of animal spirits, and suggests that each division of intellect may have a special organ in the brain. The earliest systematic attempt to localise cerebral function was, however, made by Gall and Spurzheim in the first quarter of the nineteenth century. Though their system of phrenology was wrong and was discredited, yet the criticism it called forth did much to encourage topographical localisation. In 1822, the important discovery was made, first by Majendie and later by Sir C. Bell, of the motor nature of the anterior and sensory nature of the posterior nerve roots, and about the same time it became established that aphasia is connected with lesions of the frontal lobes. In 1836, Marc Dux reported a series of 140 cases demonstrating the connection between right-sided hemiplegia and aphasia; and in 1861 this fact was elaborated by Broca, who made the first definite discovery in cerebral localisation, tracing aphasia to lesion of the left inferior frontal convolution. He was later followed by Hughlings Jackson's observations as to localised epileptic convulsions, and by the experimental and clinical work of a host of other observers.

JOHN D. COMRIE.

**HEAT CONTRACTION IN NERVE.** BRODIE and HALLIBURTON,  
(8) *Journ. Physiol.*, Vol. xxxi., No. 6, p. 473.

HALLIBURTON had previously separated three proteids from saline extracts of mammalian nervous tissue by fractional heat coagulation,—neuro-globulin  $\alpha$ —coagulating at 47° C., a nucleo-proteid containing 0.5 per cent. phosphorus coagulating at 56° C., and neuro-globulin  $\beta$  with a coagulation point of 70°–75° C.

In the present investigation, using the same method, the authors have been able to separate out from saline extracts of the brain and cord of the pigeon, three proteids coagulating at 50°–51° C., 58°–60° C., and 75°–77° C. respectively. In the case of the frog they found four proteids, the temperatures at which the coagula separated out being 39°–40° C., 47° C., 59°–60° C., and 72° C. respectively. In each case the opalescence of the solution begins to increase a few degrees below the temperature stated, and if this lower temperature is maintained long enough, that particular proteid will be completely coagulated.

They next turned their attention to the question of heat contraction in nerve, and the results obtained are very interesting. A freshly isolated frog's sciatic nerve was laid on a trough of mercury, covered with normal saline, and the whole placed in a water bath. One end of the nerve was fixed to the trough, and the other attached to a light aluminium lever carrying a mirror which reflected a spot of light on to a graduated scale, and in this way they were able to measure the amount of shortening of the nerve. On warming the water bath gradually, the first contraction began slowly when the temperature had reached 36° C., and increased until a little over 40° C. After this the length of the nerve remained constant until 46° C. was reached, when it began to shorten slowly once more, and the contraction increased rapidly up till 50° C. This second contraction was the most extensive. There was a further slight shortening at 62°–63° C. due to the connective tissue, and finally a greater contraction at 70°–75° C. Experiments on the frog's spinal cord gave contractions at temperatures exactly corresponding to those obtained with the nerves.

In the case of mammalian nerve tissue (rabbit and cat), the first shortening took place at 43°–49° C., and the second at 56°–58° C., and in birds (pigeons) at 47°–53° C. and 56°–60° C. respectively. A further shortening was observed at 62°–64° C., but this was due to the contraction of the connective tissue.

On comparing now the results of the heat coagulation of extracts with those of the heat contraction of nerve and spinal cord, the authors conclude that each shortening is due to the



coagulation of a nerve proteid which separates out at that particular temperature. The first proteid is the most important, for the death temperature of the nerve is the temperature at which this proteid coagulates. In the frog this occurs at 39°–40° C., in the mammal 47°–49° C., and in the bird 50°–53° C. A small amount of slow contraction occurs a few degrees below those just mentioned, and this corresponds to the stage of opalescence when the saline extract is gradually heated. This lower temperature, then, is the death temperature of the nerve tissue if it be subjected long enough to it. "These facts indicate a biological adaptation of the tissue proteids of animals in relation to their normal temperatures and to the rise of temperature to which they may be safely subjected," and they also probably explain the cause of death from hyperpyrexia.

SUTHERLAND SIMPSON.

### PSYCHOLOGY.

**ON THE CLINICAL MEASUREMENT OF FATIGUE.** (Ueber (9) *klinische Ermüdungsmessungen.*) Part I. "The Measurement of Mental Fatigue" (with 24 figures). By W. SPECHT, *Arch. f. d. Gesamte Psychologie*, Band iii., Heft 3, 1904.

AFTER some preliminary remarks on the rôle of fatigue in the development of the mind, the author reviews the various clinical methods that have been used to estimate fatigue. Measurements made by the esthesiometer and ergograph give inexact results, as other influences than fatigue come into play, and are difficult to estimate. Specht has adopted the method of continuous work used by Kraepelin for the same purpose. This consists in the continuous addition of columns of single figures. By comparing the number of figures added each minute, one can chart a work-curve of the individual. Specht made his patients add for five minutes, and then after a pause of five minutes, add for a second period of five minutes. On alternate days he made the patients add ten minutes consecutively. By comparing the amount done in the first minute after the pause with that done during the second minute of the first period (the second minute is chosen in order to eliminate the initial exaggeration due to released tension), one finds an increase of  $x$  due to the practice in the exercise, the fatigue having been eliminated by the pause. If this improvement through practice were not counterbalanced by the fatigue the second period of five minutes would show more work than the first period of five minutes, even if no pause were to intervene; but there is a diminution instead of an increase of work done, and

the proportion of this diminution to the work done in the first period plus the gain through practice gives Specht his fatigue-coefficient. The figure must be taken in conjunction with the work-curve, which enables us to modify it.

Out of six cases of traumatic psychoses, an extreme degree of fatigue was determined by this method in four cases; in two the output was so small that the degree of fatigue could not be estimated. In the patients with great fatigue, the restorative action of the pause was much less than in normal individuals. Simulation is at once detected by this method, as an appropriate work-curve is too complex to be produced voluntarily.

Obviously the method needs full co-operation of the patient, and the author does not discuss the point whether his exact figures yield much more than careful clinical observation of the patient.

C. MACFIE CAMPBELL.

#### HOW SHOULD WE MEASURE THE FATIGUE OF SCHOOL

- (10) **CHILDREN ?** (*Comment doit-on mesurer la fatigue des écoliers ?*) M. M. C. SCHUYTEN, *Arch. de Psychol.*, T. iv., No. 14, 1904, p. 113.

THIS paper deals with one special question regarding the fatigue of school children—namely, Are the afternoon hours worth more or less than the morning hours from a teaching point of view ?

Everyone takes for granted that the afternoon hours are not so valuable, but, as M. Schuyten points out, if this is the case, it is desirable to have scientific demonstration of it, and it is also desirable that the results gained by different scientific methods should be shown to agree with one another. At present this is far from being the case.

The methods in use may be classified under three heads: (a) psychological; (b) psycho-physiological; (c) pathological. Of these the first, which consists in setting some form of intellectual test (such as dictation or simple calculations) to the scholars, and estimating their fatigue by the comparative number of mistakes made at different hours, has led to most contradictory results. The second method, which consists in testing the children with the esthesiometer or the ergograph at regular intervals during the day, has led to more satisfactory results, in so far as all the experimenters agree that their figures show a diminution of the scholars' power in the afternoon; but when they go farther and seek to arrange the different lessons in order according to the amount of fatigue they provoke, the tables of studies which they draw up present only the vaguest resemblances one to another.

On the other hand, physicians, to whom belongs the third method, are united in their condemnation of afternoon classes.

This being the state of matters, M. Schuyten has carried through a new series of experiments devised to test the first two methods, and to see whether a reason cannot be found for the conflicting results arrived at. Three sets of experiments—one psychological, one with the dynamometer, and one with the esthesiometer, are described, and the figures obtained given.

The general conclusions to which the writer comes are these: (1) The psychological methods are of such extreme delicacy owing to the complexity of the conditions, and in using them it is so easy to obtain just the results that one wishes, that up to the present little can be said to have been demonstrated by them. By his researches of 1896 and 1897, M. Schuyten believes himself to have shown that the child's power of voluntary attention is indubitably less in the afternoon than in the morning, and this conclusion has been since confirmed by other observers; but, as he points out, this is far from implying that all afternoon instruction is inferior to that of the morning, for the simple reason that a child has other faculties which must be trained, and these faculties have not yet been tested. (2) Of the psycho-physiological methods, by far the most satisfactory is the esthesiometrical, for this method alone discounts the factor of *interest* which dominates the child's being, and has an incalculable effect upon the results obtained. Its only requirement is that the child should be attentive, or should wish to be so. • It is the only method which has emerged victorious from the test experiments which M. Schuyten has devised and carried out.

The recognition which M. Schuyten accords in this paper to the emotional element—to hope, fear, curiosity, above all to interest—certainly marks a distinct advance in the mode of investigation of the problem under consideration. Those investigators who set a class of children every hour such a task as copying unconnected letters placed upon a board, and expect their results to indicate anything but the change from the eager interest of a new game to the listless ennui of a stupid prescribed task, seem to us to have little knowledge of child nature. The children described by M. Schuyten who, after two weeks of daily ergographic exercises, have come “to submit docilely, with scepticism and irony if they are intelligent, with nothing in their mind or with disgust if they are stupid,” seem to us the most human little mortals we have met with in the records of such experiments.

The whole question of the relation of the afternoon hours to the morning hours is certainly one of interest, but it is not one of supreme importance. The important question is, Would it be

more healthful for the child to have his school hours shortened? —that is, is he likely to be able to employ himself in the afternoon more healthfully at home than at school? He will certainly not give his brain absolute rest, for many of his games make more demands upon it than much of his school work. In all his waking hours he must be doing something, and in many, if not in most cases, it seems that, even if he is not at his best in the afternoon, he may be quite as healthfully employed in school as at home.

MARGARET DRUMMOND.

**OBSERVATIONS UPON THE INTERNAL LANGUAGE (ENDO-  
(11) PHASIA) OF CHILDREN.** (*Observations sur le langage intérieur des enfants.*) AUGUSTE LEMAITRE, *Arch. de Psychol.*, Aug. 1904, p. 1.

It can no longer be doubted, says M. Lemaitre, that according to the type to which one belongs, one *hears* one's thought, or *reads* it, or *articulates* it, whether one resorts exclusively to one of these means, or combines them diversely. A couple of years ago, M. Lemaitre, who had been studying this subject in children, published the results of his observations upon 14 cases (*Le Langage intérieur des Enfants*, Lausanne, 1902, out of print), and now returns to the subject in a lengthy paper in which he summarises his previous work, and describes in detail a number of other examples, the old and the new together amounting to 32. He has also studied other cases which are not detailed, but which are included in his statistics.

His cases seem to show that in children of about 13 or 14 years of age one meets with the most diverse types of endophasia, and even that these types, when closely examined, are more complex than in the adult, in whom one centre gradually gains predominance over another. This tendency to simplification as age advances has even been observed by the writer in particular instances. For example, in 1902 he had in a class of 31 scholars, 3 of the visual-auditive type. Two years later the type was unchanged in 2 instances, but in one it has altered to pure audition with secondary visualisation.

Out of a total of 90 pupils in three consecutive classes, and whose average age was 13 to 14 years, M. Lemaitre obtained the following result:—

Varieties.	Number.	Percentage.
Verbo-moteurs . . .	41	45·55
Verbo-auditifs . . .	12	13·33
Verbo-visuels . . .	14	15·56

Varieties.	Number.	Percentage.
Symbolo-visuels.	15	16·67
Auditivo-visuels	5	5·56
Visuéo-moteurs	2	2·22
Équilibrés	1	1·11

Differences in the type of mentalisation may perhaps, says M. Lemaitre, account for some of the controversies with which philosophers have wearied the world. For example, in the famous scholastic dispute concerning the nature of Universals, the nominalists, the conceptualists and the realists may have had different forms of endophasia, belonging respectively to the motor, to the auditive, and to the visual type. Some confirmation of this idea may be found in the manner in which pupils of 13 or 14 years of age represent abstract ideas in accordance with the type to which they belong. Some take hold of an abstract idea with great difficulty; others, and especially those of the visual type, apprehend them easily, but only by instinctively transforming them into concrete notions.

For example, the idea of the Infinite was suggested to a number of pupils. Of these, 2 of the motor type said: one that the infinite was inconceivable, and one that it was impossible and must have an end sooner or later; of 5 visuals, 2 saw the starry sky, 2 a closed circle, and one a black point on a white ground; an auditive said that the thought of the infinite produced a disagreeable sensation, a feeling of breathlessness, of oppression, while at the same time he seemed to be running and leaping and chasing away the mists which enveloped him.

As M. Lemaitre's paper is very largely a record of cases individually detailed, it should be studied *in extenso*. The various types which the author recognises in the above table are not defined, but the reader may follow what is meant by studying the cases cited. An investigation on similar lines of a group of British children would be a most interesting piece of work for anyone who has the opportunity.

W. B. DRUMMOND.

## PATHOLOGY.

### THE CEREBRO-SPINAL FLUID IN RELATION TO DISEASES

(12) OF THE NERVOUS SYSTEM. F. W. MOTT, *Brit. Med. Journ.*, 1904, p. 1554.

DR MOTT points out that the cerebro-spinal fluid resembles in composition the amniotic fluid and the sweat, and that true albumin and fibrinogen are absent, it is therefore a secretion

and not an exudation. The fluid probably secreted by the choroid plexus passes into the subarachnoid space through the foramen of Majendie, and at each cardiac systole is driven from the cranium into the spinal canal.

#### SOURCE, DESTINATION AND FUNCTIONS.

The epithelium covering the choroid plexuses consists of polygonal cells with large nuclei, their cytoplasm containing a number of round and oval clear areas, probably due to the contained secretion. Clinical observations show that the fluid is continually being secreted, and its amount has been estimated as in the well-known case of St Clair Thomson. The fluid thus continually secreted fills up all the spaces, cracks and crevices, in the subarachnoid space, and a quantity is as continually escaping. It may escape along perineural lymphatics, and these may be avenues of infection in the production of meningitis. Cushing, however, has brought forward experimental evidence to show that the fluid chiefly finds its exit into the longitudinal sinus.

A layer of arachnoid like a sleeve follows the pial vessels as they dip into the substance of the brain, and thus forms an important canicular system—the perivascular lymphatic system—and the column of blood in the thin-walled vessels is thus surrounded by a constant fluid pressure.

There is little tendency for *lymph* to pass from the capillaries into the canicular system, and Mott has never seen signs of coagulated proteid in this system. How is the brain nourished? First, since in starvation the brain does not waste, its metabolism is small but complex, choline and acid are constantly formed, thus Cavazzari states that the alkalinity of the cerebro-spinal fluid is just a trifle over one-half that of blood. Secondly, oxygen is certainly necessary, and Dr Mott asks, what are the oxygen and carbonic acid tensions of the cerebro-spinal fluid? He suggests that the oxygen is largely required in promoting the bio-chemical changes in the synapses of the neurons. Dr Mott then points out that the neurons differ much from other cells of the body in their complexity, their similar histological structure but diverse functional activity, and in their incapacity for regeneration. Regeneration is incompatible with the differentiation of function which has been acquired by long ages of biological evolution. Sicard maintains that there is a true vascular lymphatic sheath enclosed by the cerebro-spinal fluid, and that this fluid only exists round the arteries, and does not extend to the capillaries, and Mott has seen in sleeping sickness a delicate sheath containing lymphocytes immediately surrounding the vessel, and outside this the canicular system. Perhaps the cerebro-spinal fluid is itself

the medium of gaseous exchange between blood and tissue, into which the chemical products of nervous activity pass.

**EFFECTS OF INCREASED INTRACRANIAL PRESSURE FROM INTERFERENCE WITH ESCAPE OF THE FLUID FROM ITS SOURCE IN THE LATERAL VENTRICLE.**

Such a condition produces hydrocephalus, and Mott refers to three cases of slowly growing non-malignant tumours of the third ventricle, and one of chronic basal meningitis caused by caries of the petrous bone. They all showed signs of increased intracranial pressure, vomiting, headache, optic neuritis, tremors, fits, drowsy stupor with progressive mental enfeeblement. He mentions one of these cases which had been under his own care at Charing Cross Hospital. A tumour blocked the iter and led to distension of the third and lateral ventricles; there was a flattening of the convolutions, due to cortical capillary anæmia and venous stasis, and microscopically there was shown to be dilatation of the perivascular canalicular system. The symptoms came and went for no other reason than the change in position of the tumour in obstructing the iter. The upright position, and therefore gravity, favoured the onset of the symptoms, whilst the prone position aided the recovery to the normal state of consciousness.

**ABNORMAL CHEMICAL CHANGES IN THE FLUID.**

Is the pia arachnoid an impermeable membrane? Sicard maintains that it is so, but morphia, potassium iodide, and cocaine injected into the subarachnoid space all produce toxic symptoms. In uræmia, urea is present, and sugar in diabetes—yet the variations found in pathological conditions are slight, and favour the idea that the fluid is a special secretion which prevents these substances passing through. Poisons act on the nervous system probably by osmosis from the blood into the canalicular system. Donath has shown that, as originally stated by Mott and Halliburton, choline can be found in the cerebro-spinal fluid in a large number of organic nervous diseases.

**ORGANISED ELEMENTS IN THE CEREBRO-SPINAL FLUID.**

**(a) LEUCOCYTES; (b) MICRO-ORGANISMS.**

The centrifuged normal fluid contains no organised elements, but leucocytosis is one of the earliest signs of organic disease of the nervous system. The acute diseases are associated with polynuclears, the chronic with lymphocytes. When the membranes are affected in diseases of the nervous system, the mononuclears are seen; in functional psychoses and neuritis there is no lympho-

cytosis. Mott considers that the leucocytosis is in response to the escape of products of degeneration into the cerebro-spinal fluid. That polynuclears are phagocytic is well known, but Ehrlich has denied that the lymphocytes are capable of amœboid movements. In sleeping sickness, however, when all the perivascular canalicules are filled with mononuclears, Dr Mott has seen these elements on their way through the walls of the vessels. The endothelial cells lining the subarachnoid space are also phagocytic, and the existence of these macrophages containing red blood corpuscles would certainly lead to the diagnosis of hæmorrhage.

Micro-organisms are in disease rarely found in the cerebro-spinal fluid, but are in cerebro-spinal and tuberculous meningitis. Schiff concludes that the nose is the seat of the infection, and that the diplococcus of Weichselbaum is not infrequently present in the human organism; and Flatau has shown that there are perineural and perivascular lymphatic connections between the nasopharynx and the subarachnoid space. Infection, Mott thinks takes place in this way, and not by means of migrating leucocytes containing these micro-organisms.

#### SLEEPING SICKNESS AND INFECTION OF CEREBRO-SPINAL FLUID BY TRYPANOSOMES.

Mott first showed that the pathological change in the sleeping sickness was a meningo-encephalo-myelitis, and Bruce has found the trypanosome to be constantly present in the cerebro-spinal fluid. Is the trypanosome the cause *per se*, or does it lead to a secondary infection as a carrier of micro-organisms? Castellani and Mott have found a diplococcus present in the blood, whereas it is remarkable that the trypanosome is rarely found in the blood or in the perivascular cell infiltration of the central nervous system. Mott is of opinion that the lethargy is due to cerebral anæmia, caused by compression of the small vessels by the accumulation of lymphocytes in the perivascular spaces. The tremors, paresis and fits are the result of irritation phenomena associated with stasis in the vessels and degeneration changes in the neurons and neuroglial proliferation. Mott states that experiments on monkeys do not afford any conclusive evidence of the trypanosome being the cause of the disease, but regards the etiological evidence as being convincing; he suggests that there may be some plasmodial or transitional form of the parasite not yet demonstrable in sections of the tissues.

#### GENERAL PARALYSIS.

Mott alludes to the occurrence of plasma cells in the disease. He regards them as altered lymphocytes, and though always



present in general paralysis and tabo-paralysis, are not pathognomonic, and states as a significant fact that he has seldom found diplococci in this form of chronic meningo-encephalitis, whereas diplococcal infection is extremely common in sleeping sickness.

W. B. WARRINGTON.

[In Lipæmia.—Dr E. E. Glynn and Dr Roaf gave an account of a case of lipæmia at the Liverpool Medical Institution on December 8th. The cerebro-spinal fluid had the appearance of milk. Its sp. gr. was 1014, and it contained 2·2 per cent. of proteid, 1·56 per cent. of fat. Sugar not estimated, but present.—W. B. W.]

#### DO THE PARATHYROID GLANDS PLAY A RÔLE IN HUMAN

(13) **PATHOLOGY?** (*Spielen die Glandulæ parathyroideæ in der menschlichen Pathologie eine Rolle?*) HERMAN LUNDBORG, *Deutsch. Ztsch. f. Nervenheilk.*, Bd. 27, Heft 3-4, 1904, p. 217.

THE author reviews the work already done on the thyroid gland system, quoting from Sandström, Jeandelize, Biedl, and many others. Experimental results seem to show that the parathyroids are of considerable importance, and the assumption is that they must play an important part in human pathology. Lundborg thinks that myotonia congenita is a disease due to a hypoparathyroidea. Tetany and this affection sometimes appear together and have many points in common, but whereas tetany is believed to be due to an intoxication, myotonia has been regarded as being dependent upon a congenital condition of the muscles. The author thinks that myoclonus occupies a position midway between these two diseases. He has seen myoclonus and paralysis agitans combined in the same patient, and believes that there is a relationship between these two conditions. Paralysis agitans is, in the author's opinion, to be interpreted as a chronic progressive hypoparathyroidea. According to this view, then, paralysis agitans is a disease analogous to myxœdema, and has its origin, in the first instance, in a slowly advancing hypothyroidea. Both diseases appear usually after forty years of age, and develop gradually; both end in cachexia; both may occur simultaneously in the same individual (Lundborg). In cases of advanced myxœdema, symptoms sometimes develop like those due to a hypoparathyroidea—tremor, tetany, clonic twitchings, and epileptiform attacks. Luzzatto's case of paralysis agitans with myxœdema is quoted at length, and is regarded as a thyroid, afterwards followed by a parathyroid insufficiency. Dana and Möbius hold similar views

on this question, and Frenkel in 1899 noted that the skin and subcutaneous tissue was thickened in patients suffering from paralysis agitans.

If, then, paralysis agitans is a disease allied to myxoedema, resting upon a chronic progressive hypoparathyroidea, is there, asks Lundborg, in human pathology a disease which stands in the same relation to paralysis agitans as Graves' disease does to myxoedema—that is, a disease which is, to some extent, a marked contrast to paralysis agitans? He thinks this disease is myasthenia gravis pseudo-paralytica (Jolly). This affection shows a striking contrast to paralysis agitans, and it is not infrequently combined with Graves' disease, or with certain symptoms of that disease. If the foregoing chain of induction is correct, one must regard the parathyroid as a regulating organ, whose function is to maintain undisturbed muscular (or neuro-muscular) activity.

Lundborg puts his hypotheses into diagrammatic form, and emphasises the fact that although he holds these diseases to be more or less related to each other, and probably to be dependent upon auto-intoxication, yet it must be regarded as quite unsettled whether or not the parathyroid glands play a definite part in their pathogenesis.

OLIPHANT NICHOLSON.

#### INFLUENCE OF ALCOHOLISM ON THE REPRODUCTIVE

(14) POWERS AND ON THE OFFSPRING. (*Influenza dell'alcoolismo sul potere di procreare e sui discendenti.*) CARLO CENI, *Riv. Speriment. di Freniat.*, 1904, Vol. xxx., f. ii.-iii., p. 339.

THE influence of alcoholism as one of the factors in the deterioration and degeneration of the race is a question of perennial interest.

Attention has been called, especially in France, to the relation between alcoholism and the fertility of a nation, but authorities differ in their interpretation of statistics on the subject. Some consider alcoholism the principal cause in the depopulation of France, but Arrivé contends that the lowered birth-rate in that country is to be explained by other factors. Sonilhé even goes so far as to maintain that alcohol increases the number of births.

Numerous clinical and statistical observations from many countries are at one in bearing testimony to the disastrous effects of alcoholism on the offspring, showing clearly the always high proportion of idiots, imbeciles, and neuropathics in general, specially epileptics, in the families of alcoholics.

Some authorities do not admit that alcoholism alone in the parents, without the co-operation of some other factor, more

especially tuberculosis and a nervous heredity, can be the cause of idiocy, etc., in the children.

Numerous experiments by Mairet, Carrara, and Martinotti, undertaken to show the direct action of alcohol on the embryo during its development, cannot be accepted without some reserve, inasmuch as it is not possible to exclude absolutely that the results may be due in some degree to an indirect action of the alcohol, which may act, like any other factor, in producing alterations in the ovular appendages, and thus interfere, but only indirectly, with the nutrition of the embryo.

More grave are the objections to the experiments of Feré, on account of the power of alcohol to precipitate albuminoids. For this reason we cannot admit that the monstrosities produced by him should be considered exclusively as the result of the direct action of alcohol in the embryo.

The experiments of Ceni were made to enquire into the part played by alcoholism, specially that of a chronic nature, in the production of imperfect conceptions and of the various anomalies of development which are met with so frequently in the children of alcoholics, considered independently from the action which alcohol can exert on the embryo during maternal gestation.

Two cocks and five hens were subjected to a chronic progressive intoxication with 40 per cent. ethylic alcohol administered by the mouth, beginning with a daily dose of 1 c.c. and reaching at the end of five months a maximum dose of 15 c.c. Then after adaptation had been established, the dose was reduced to 10 c.c., this representing the maximum amount well borne by the fowls without phenomena of serious intoxication or appreciable disturbance of their general condition. One cock and two hens, however, died in the first two months of adaptation with symptoms of a subacute ulcerative gastro-enteritis.

The fowls which survived lived, in their alcoholised state, in excellent condition to all appearance for from one to two years, but then exhibited a rapid and progressive general deterioration which quickly ended in death in a state of cachexia, lasting about two months in each subject.

The autopsy showed the organs of reproduction to have almost their normal form, appearance and consistency, without any sign of evident atrophy. This point is important, and all the more so as atrophy of the reproductive organs has been a constant positive result in fowls fed with damaged maize, and those in which the thyroid had been removed. As regards the number of eggs produced by the three hens under observation, in the one year they laid a total number of 140 eggs; that is to say, an average of 46 per hen, whereas the average for a healthy fowl is 110-120.

It is thus shown that chronic alcoholic intoxication acts

directly on the organs of reproduction and is prejudicial to the fecundity of animals. Its effects in this respect, however, appear to be less than those of the toxin of Pellagra, as well as those resulting from extirpation of the thyroid gland.

Ceni does not believe that the sexual organs can really be considered as more liable to injury from alcoholic intoxication than the other organs of the body.

In order to observe the effects of chronic alcoholism on the products of conception, he incubated eggs laid by alcoholised hens and fecundated by an alcoholised cock. Of these eggs 130 were incubated for varying periods, and 10 others were brought to complete incubation, the former in an incubator, and the latter under a brood-hen. Of the 130, 100 were kept at a constant physiological temperature, while the remainder were subjected to variations.

The 130 eggs were opened after 90-100 hours of incubation, and produced 56 living embryos of normal development; that is to say, 43 per cent., including in that number even those which had reached a development corresponding to that of 40-50 hours. All the other eggs showed either arrests of development, which were complete or of high degree, or death of the embryo, or thirdly, various developmental anomalies.

The eggs of healthy hens gave 77 per cent. of normal living embryos. Ceni holds that his experiments clearly prove that the embryonic germ of alcoholised parents carries in itself a true diminution of organic resistance transmitted from the parents, as shown by the fact that it cannot tolerate variations in temperature which are well enough borne by germs in normal condition.

The author summarises his conclusions as follows :—

(1) Chronic alcoholism has a direct action on the reproductive powers, impairing the fecundity, though not in an aggravated manner.

(2) Chronic alcoholism in the parents has a disastrous influence on the progeny, producing in them a diminished organic resistance, and anomalies of development of various degrees and kinds, by the direct action exerted by this poison on the primitive sexual elements (ovum and spermatozoon) before conception.

(3) In virtue of the diminished organic resistance inherited by the germ from parents subjected to alcoholic poisoning, the least causes which can disturb the regular course of the development of the ovum are sufficient to produce arrests and grave anomalies of development in the embryo.

(4) The baneful influence of alcohol in the parents upon the progeny is made clear, at one time directly producing in them a state of general debility (arrests of development *in toto*, premature

death), and at other times determining secondarily anomalies and partial arrests of development in the embryo in consequence of primary lesions in the ovular appendages. T. C. MACKENZIE.

### CLINICAL NEUROLOGY.

**ACUTE ANTERIOR POLIOMYELITIS IN THE ADULT.** (*La polio-  
(15) myélite antérieure aiguë de l'adulte.*) A. VAN GEHUCHTEN,  
*Nevrage*, Vol. vi., f. 3, Oct. 15, 1904, p. 279.

THE earlier portion of this paper deals with an historical review of the published cases of acute poliomyelitis in the adult.

The following details of the case under consideration are then given:—A girl aged 21, at the end of June 1900, began to suffer from headache and pains in the lower extremities. The feeling of malaise slowly increased till July 5, and on that day the headache became intense, she had a rigor and was obliged to keep her bed. On July 10 the fever had gone, but when she tried to stand she fell in a mass on the floor. Some few hours later she was completely paralysed in all four limbs and in the trunk muscles.

She was admitted to hospital on July 20. There was complete flaccid paralysis of all the muscles of the arms and legs and also of the trunk muscles, except for slight power of flexion and extension of the toes on the left side. The thoracic muscles were motionless. Some of the muscles of the neck were paralysed. The tendon reflexes were abolished. The superficial reflexes were all abolished except slight flexion of the little toe and a slight abdominal movement.

Sensation was perfect to all forms.

The muscles themselves were, however, very sensitive to pressure, but direct pressure on the nerves gave rise to no pain. The bladder and rectal functions were unaffected. The patient died on Sept. 19, 1900. Macroscopically nothing abnormal was detected. Microscopically the lesion affected the grey matter of the spinal cord bilaterally from the fifth cervical segment to the second sacral. An irregular cavity existed in the anterior horn in the cervical region. In the lumbar region the anterior horn had shrunk almost to the size of the posterior horn. In the dorsal region the anterior horns had retained their normal size, but were filled with a granular substance without any trace of nerve fibre. There was a complete absence of nerve cells in the regions affected. The posterior nerve-roots were normal, whilst the anterior nerve-roots were atrophied.

The anterior horns of the affected region showed very marked vascular changes. A large number of engorged blood-vessels with distension of the perivascular space by extravasated cells were

present. In addition to the small cell infiltration of the horn there were present cells which the author considered were neuroglia cells undergoing proliferation.

The vascular lesions were present in the grey matter, whilst the vessels of the pia mater were normal.

It is noted that the arteries of median fissure appear normal up to the point where they enter the grey matter, and that the lesion affects the *veins* predominantly if not exclusively. The view is taken that the infection is carried to the cord by means of the vessels, but there are three important points observed in the present case:—

(1) That the arteries of the pia mater and anterior fissure are normal, whilst there is a marked lesion in the veins of the anterior fissure.

(2) The predominance of the vascular lesion in the grey substance of the cord and the absence of vascular lesion in the white matter.

(3) The lesions occur in the course of one vessel to the exclusion of others.

And from the above it is believed that the lesion of anterior poliomyelitis in the adult as in the child is due *not* to an affection of the arteries of the median fissure, nor to one or other of its branches, but to an affection of the capillaries of the grey matter.

The lesions found are then compared with those which occur in anterior poliomyelitis of the child, and it is concluded that the lesions are identical. One cause acts in both children and adults, viz.: *an infection*. The initial lesion is vascular and it is impossible to regard the vascular changes as secondary to changes in the cells of the anterior horn.

Why the vessels in the anterior horn are picked out is a fact which has as yet no explanation. The paper ends with a most interesting discussion on the experimental interruption of the abdominal aorta and its effect on the lumbar cord. The above digest does but incomplete justice to a most carefully worked out paper on a subject of which the morbid anatomy is now well known, but the pathology still remains obscure.

FREDERICK E. BATTEN.

## TWO CASES OF SO-CALLED POLIOMYELITIS ANTERIOR

(16) CHRONICA IN FATHER AND SON. (Zwei Fälle von sogenannten Poliomyelitis anterior chronica bei Vater und Sohn.)

J. BRUINING, *Deutsch. Ztschr. f. Nervenheilk.*, Bd. 27, 1904, p. 269.

THE elder of these two patients, a previously healthy mason, 45 years of age, without apparent exciting cause, developed weakness and muscular atrophy, commencing in the proximal muscles of

the right lower limb, and spreading distally. In three months he became unable to work. Six months from the commencement of his illness he noticed weakness of both shoulders, and the movements of the arms became weak. Here also the weakness progressed from the proximal muscles towards the periphery. After two months more, weakness appeared in the left thigh, and gradually spread below the knee. Atrophy and weakness appeared simultaneously in the affected muscles. Pains and paræsthesiæ were absent. There was no anæsthesia. The reflexes, superficial and deep, were diminished. In the course of the disease, fibrillary tremors appeared in the muscles, and in certain of them slight degenerative reactions were made out. Most muscles, however, simply showed quantitative diminution of electrical excitability. From weakness of the chest muscles a bronchitis which supervened developed into a pneumonia, and proved fatal, the total duration of the disease being  $1\frac{1}{2}$  years. Post-mortem marked atrophic changes were found in the anterior cornual cells and the anterior roots. There was no degeneration in the lateral columns, as in amyotrophic lateral sclerosis; the posterior columns were also normal. There appeared to be some diffuse affection, slight in degree, at the margin of the antero-lateral columns.

The patient's son, a coachman, 23 years of age, the eldest of a family of thirteen, developed muscular atrophy, commencing in the muscles of the neck, and progressively attacking those of the shoulder-girdles, upper arms, forearms and hands, producing in about nine months an almost total paralysis of the neck and shoulders, with marked paresis of the arms. Paralysis and muscular atrophy occurred concurrently. The thoracic muscles became affected also, but the muscles of the lower limbs remained unaffected. Sensibility was normal, and there was no tenderness of nerve-trunks. The deep reflexes in upper and lower extremities gradually disappeared. Fibrillary movements and reactions of degeneration were present in some of the affected muscles. The sphincters were unaffected. Slight bulbar symptoms appeared, in the form of subjective difficulty in swallowing, and some alteration of articulation. After about 15 months the patient died of inter-current pulmonary affection. No autopsy was obtained.

Bruining calls attention to the rapid course of the disease in these two patients, and to the unusual order in which the muscles were attacked, spreading not from the periphery upwards, but from the proximal muscles distalwards. The cases are examples of chronic poliomyelitis anterior, not inflammatory in origin, but a primary degeneration. The process was confined to the anterior cornua, Clarke's columns escaping. There was probably a hereditary weakness in the cells of the anterior cornua.

PURVES STEWART.

**PURULENT MYELITIS—FOCAL AND DISSEMINATED. J.**

(17) COLLINS (New York), *Journ. of Nerv. and Ment. Dis.*, Nov. 1904, p. 695.

COLLINS gives the clinical observation and pathological findings of this case. Patient was an unmarried Frenchwoman, aged 25, who shortly after an attack of influenza, in January 1901, complained of paræsthesia and pain in the left leg and back. Until June he was able to walk with aid of a stick; she then received vigorous antisyphilitic treatment, but got worse; greater feebleness and pain, especially in front of left thigh and groin. In January 1902 the right lower extremity became involved. In February she presented exaggeration of both knee-jerks, patella and ankle clonus; Babinski reflex on both sides; anæsthesia on the anterior surface of the left thigh; anæsthesia and thermo-anæsthesia on the outer side of the same area; less disturbance on the posterior surface. Right thigh completely insensitive save on anterior surface. In April the symptoms were: complete paraplegia; loss of plantar reflexes; patellar and Achilles reflexes present. Anæsthesia, thermo-anæsthesia, and some analgesia from the level of the iliac crests downwards. The autopsy showed an extensive disseminated purulent myelitis with numerous foci and one relatively large pus cavity in the sacral region forming the primary focus, to judge by the symptoms.

C. MACFIE CAMPBELL.

**A CASE OF DISLOCATION OF THE ATLAS. J. H. LLOYD,**

(18) (Philadelphia), *Amer. Journ. of the Med. Sci.*, Nov. 1904, p. 751.

PATIENT, a man aged twenty-one years, on the 16th September 1903, fell off a cart on his left shoulder, the cart-wheel passing over the back of his neck. From this date he could not rotate the head without moving the shoulders. On 12th January 1904, he slipped and fell on his left shoulder, striking the back of his head on the pavement. On examination after a short stay in a surgical ward, patient presented a marked deformity in the neck most prominent on the left side: the head was slightly twisted to the right; a finger in the pharynx palpated a projecting mass on the posterior wall. On the left side the upper arm was paralysed; he could not pronate nor supinate forearm; could not flex middle ring or little finger, nor separate the fingers. No atrophy of the muscles; slight diminution to faradism in deltoid and pectoral. No other muscle groups were affected. Sensory symptoms: loss of thermic sense in the right arm and forearm, diminution of it on the whole right side from the level of the fourth cervical vertebra. Above this there was a narrow band of hyperæsthesia. No tactile



anæsthesia anywhere save over the distribution of the great occipital nerve of the left side. This last symptom and the skiagram of the case led to the diagnosis of dislocation of the atlas. The sensory symptoms cleared up, the motor affection persisted. Lloyd has collected several other cases of dislocation of the atlas which did not prove fatal.

C. MACFIE CAMPBELL.

**A CASE OF TUBERCULOUS MENINGITIS WITH SECONDARY**

(19) **INFECTION.** S. S. KNEASS and J. SAILER, *Journ. Nerv. and Ment. Dis.*, Oct. 1904, p. 660.

NOTES are given of a case in which the autopsy disclosed acute disseminated tuberculosis of lung, spleen, liver and kidney, with early acute tuberculous meningitis: coverslips from the meninges stained for tubercle bacilli showed them present in large numbers.

*Intra vitam* the predominant symptoms were those of severe septic infection, involving the cerebral hemispheres, and the existence of malignant endocarditis was suspected at first: the symptoms of cerebral meningitis, with very slight signs of spinal involvement, were fully developed. Kernig's sign was present, and "our experience leads us to agree with Dieulafoy and Packard that it is rare in tuberculous meningitis, but not so uncommon as to make its presence of especial significance."

Lumbar puncture was performed twice, and yielded on both occasions a clear colourless fluid, with a pronounced albumen reaction and giving the copper test for pyrocatechin: cultures were made on various media, and the same micro-organism was found on each occasion, corresponding in all cultural and morphological respects to the micrococcus tetragenous. It was therefore supposed, *intra vitam*, that the case was one of meningitis produced by this micro-organism. No inoculations could be made with the spinal fluid, as animals were not available, but smears, stained for tubercle bacilli, were negative.

The authors believe that there is no reasonable doubt of the double infection in this case. They find from an examination of literature that tuberculous meningitis, complicated by a secondary infection, is either very rare or rarely recognised. Doubt has been thrown by Stadelman on the occurrence of the meningococcus and the tubercle bacillus together, but Lenhartz and Heubner found them associated in three cases of cerebro-spinal meningitis, and Holdheim found the meningococcus in the spinal fluid of a case which proved at the autopsy to be one of tuberculous meningitis. Heiman found the tubercle bacillus and the diplococcus of pneu-

monia in the exudate of a case of meningitis. This appears to exhaust the literature on the subject. There is not enough material to show whether a difference can be recognised in the clinical pictures of cases of tuberculous meningitis with mixed infection and those without it. A. W. MACKINTOSH.

**ON CASES OF THE SYMPTOM-COMPLEX "CEREBRAL TUMOUR"**

**(20) TERMINATING IN RECOVERY. ON FATAL CASES OF "PSEUDO-CEREBRAL TUMOUR," WITH AUTOPSY.**

[Ueber Fälle von symptomkomplex "Tumor cerebri" mit Ausgang in Heilung (Pseudo-Tumor cerebri). Ueber letal verlaufene Fälle von "Pseudo-Tumor cerebri" mit Sektionsbefund.] M. NONNE, *Deutsch. Ztschr. f. Nervenheilk.*, Bd. 27, 1904, p. 169.

Now and then we meet with clinical cases in which all the usual signs and symptoms are present which justify us in diagnosing cerebral tumour, yet in which the subsequent course shows that the diagnosis was wrong, but without showing how any other diagnosis was possible. These include not only cases in which permanent cure resulted, but also those which come to an autopsy, and show entirely negative appearances.

Nonne gives notes of no fewer than 12 such cases. In most of them the classic combination of headache, optic neuritis, and vomiting were present, with focal symptoms pointing to various parts of the brain. Nonne believes that syphilis, tubercle, disseminated sclerosis, hydrocephalus, uræmia, etc., could all be excluded. We observe, however, that in case 5 the patient, who had transient amblyopia in addition to headache, optic neuritis, facial weakness, and reeling gait, subsequently developed a degree of optic atrophy, so that disseminated sclerosis may perhaps have been the underlying affection. Remissions of long duration are familiar to most who have studied this disease. In case 6, the patient, a young woman of 20, had all the ordinary signs pointing to a cortical tumour in the face and arm centres. She was actually trephined. Nothing abnormal was found, and the patient recovered.

In cases 9, 10, 11 and 12 the patients died, and in all of these (except case 9) an autopsy was obtained, and nothing was found. Case 9 apparently recovered, but died suddenly about 1½ years after her discharge. (In this case slight optic neuritis was still present when the patient was last examined.)

In addition to the 12 cases where tumor cerebri was diagnosed, he records 6 cases of hydrocephalus. Of these, two show the impossibility of diagnosing between hydrocephalus and tumour

of the posterior fossa. In one, the autopsy showed marked hydrocephalus and nothing more; in the other there was a sarcoma growing from the floor of the fourth ventricle and compressing the cerebellum and the occipital lobes. Three other cases were examples of traumatic hydrocephalus, simulating tumours of the base. In one case the hydrocephalus was confirmed by autopsy; in the other two, lumbar puncture showed enormous increase of intraspinal pressure. One of these cases was cured; the other died, and hydrocephalus was the only abnormality found post-mortem. The last case of the series was one of occipital headache, vomiting, double optic neuritis, and attacks of giddiness, with tenderness on percussion over the region of the left mastoid process, and fulness of the neck in the neighbourhood. Slight right facial weakness was present, and hearing in the left ear was somewhat deficient. There was a history of a subacute otitis media two years before. The alternative diagnosis was made of intracerebellar abscess or thrombosis of the lateral sinus. The latter condition was actually found on operation. For some time afterwards the patient showed marked unsteadiness of gait, but ultimately recovered with slight optic atrophy. Nonne attributes these symptoms to a secondary hydrocephalus in the posterior fossa.

This remarkable series is of great interest, and amplifies what must have been the occasional experience of most neurologists.

Cases like the above must modify the absolutely unfavourable prognosis which we frequently are inclined to give, in inoperable intracranial tumours. Moreover, cases which are trephined, and subsequently improve, are not necessarily to be put down as cases of cerebral tumour "cured" by such operation.

PURVES STEWART.

**A CASE IN WHICH THERE WAS A LINEAR LESION  
(21) LIMITED TO THE WHITE MATTER OF THE RIGHT  
ASCENDING FRONTAL CONVOLUTION-HEMIPLEGIA,  
WITH A STUDY OF THE SECONDARY PYRAMIDAL  
DEGENERATION. (Un cas de lésion linéaire limitée à la  
substance blanche de la frontale ascendante droite dans la  
moitié supérieure. Hémiplegie. Étude de la dégénération  
secondaire pyramidale.) PIERRE MARIE and IDELSOHN, *Rev.  
Neurol.*, Oct. 30, 1904, p. 1025.**

THE subject of this investigation was a painter, æt. 49. He had suffered from lead paralysis eight years before the hemiplegia came on, and had practically recovered. In June 1903 he was suddenly paralysed down the left side, but did not lose conscious-

ness. The power gradually returned, but in July he had a series of Jacksonian convulsions which commenced on the left side of the face and affected chiefly the left side of the body. There was also, after this attack, anæsthesia on the left side. During August and September he had several more fits, and for a week he was in a condition of mental elation. In October his mental condition became normal, but he died in the end of December. The pathological findings were as follows:—A linear scar extended in the white substance of the ascending frontal convolution from about the level of the middle of the Rolandic fissure upwards and forwards but did not involve the grey matter. The scar was clean cut as if the lesion had been produced by a knife, the lips were not adherent, and its surfaces were covered with a thin layer of hæmatoidin. By Marchi's method some diffuse degeneration was found in the white substance of the frontal region, distant from the lesion. The pyramidal degeneration was specially noted by the authors. On horizontal section in the thalamic, sub-thalamic and posterior commissural regions degeneration was found in the region of the median fillet, the anterior quadrigeminal body and in the retro-lenticular segment of the internal capsule. There was a perfectly limited degeneration of the vertical fibres in the internal capsule, the largest of those degenerated bundles being found immediately in front of Türck's bundle, and the whole of the degenerated fibres did not occupy more than a sixteenth part of the internal capsule lying anterior to Türck's bundle. At the level of the foot of the cerebral peduncle in a section passing through the red nucleus, the degeneration was crescent-shaped with its convexity outwards and its anterior end at the periphery. The convex portion constituted the boundary between the motor tract and Türck's bundle. In the bulbar region the degeneration was diffused all over the pyramidal cone.

T. GRAINGER STEWART.

**A CASE OF UNCOMPLICATED HYSTERIA IN THE MALE,**  
 (22) **LASTING THIRTY YEARS, WITH POST-MORTEM EX-**  
**AMINATION.** S. WEIR MITCHELL and W. G. SPILLER,  
*Journ. of Nerv. and Ment. Dis.*, Oct. 1904, p. 625.

THE purpose of this paper is to give "the result of minute pathological study of an uncomplicated and lasting case of undoubted hysteria." The conditions were ideally perfect: the patient had been for nearly thirty years under close observation, he had no intercurrent malady, he died suddenly from heart failure at the age of sixty.

Apart from the remarkable condition of his left arm and mental peculiarities typical of hysteria, he had very few symptoms. All the muscle reflexes were excessive, but there was no clonus; occasionally retention of urine occurred for a month or so; early in his illness he had attacks of temporary weakness of the legs.

The outstanding feature of the case was involuntary movement of the left arm, either of the nature of "pendulum" spasms or rotatory movements. (Later the right arm was affected also with spasms.) The movements ceased during sleep, and also when he lay down with his back and head flat on the ground: he could then execute with the hand the most exact and voluntary movements. If, however, he raised his head, the arm movement began and did not cease until he stood up and lay down again. The movement was exaggerated by walking, by voluntary movements of other limbs, by the act of speaking or eating, by volitional effort at control, by excitement, emotion, etc. He had power to stop the spasms by certain manœuvres, but the result was very unpleasant—"he tottered, his face became convulsed, there was horrible pain in the back of the head, there was a general convulsive movement of the entire body." Restraint of the moving arm by an observer caused "a general convulsion, involving in succession the other arm, face, neck and legs."

All kinds of treatment, sedulously employed, failed except hypnotic suggestion, which effected temporary improvement.

Minute examination of the brain and spinal cord revealed "nothing that could be regarded as distinctly pathological." The heart was the only organ which was plainly diseased, and it presented some unusual conditions.

The interest of the case is twofold—(1) The conditions for pathological examination of an undoubted case of long-continued uncomplicated hysteria were unique, and the conclusion seems warranted that "with our present methods the most typical hysteria, lasting for years, presents no sign of representative organic lesion." (2) A striking feature was the fact that "certain positions, passively caused or voluntarily produced, gave rise to definite extension of the spasms to the quieter arm, and at times to the entire body." The increase and spread of spasms by restraint of the primary spasm was very marked: Dr Mitchell has seen several cases illustrating this fact, and the writers think it probable that it may be found to occur in cases of Jacksonian epilepsy—the muscular convulsion appearing to act as a release to excessive energy stored up in the cortex. It is known that restraint of the spasms in idiopathic epilepsy is sometimes followed by sensations which are more unpleasant than are the spasms, *e.g.* giddiness and distress (Gowers).

A. W. MACKINTOSH.

**TETANY, PSEUDO-TETANY, AND THEIR TRANSITION FORMS**

- (23) **IN HYSTERIA.** (*Tétanie, Pseudotétanie und ihre Mischformen bei Hysterie.*) CURSCHMANN, *Deutsch. Ztschr. f. Nervenheilk.*, Bd. 27, 1904, p. 239.

SOME of the symptoms hitherto described as pathognomonic of true tetany, *e.g.* Chvostek's facial phenomenon, the mechanical hyperexcitability of the motor nerves, and Trousseau's symptom, are, in the author's opinion, not alone sufficient to justify the diagnosis of tetany, as they may also occur in hysterical cases (pseudo-tetany). He regards the increase of electric excitability of the motor nerves (Erb's phenomenon) as the cardinal differential diagnostic symptom between true and false tetany, as it is invariably absent in the latter condition. The author gives in detail a set of five cases from which he deduces these conclusions, and he also gives a brief summary of eight analogous cases recorded by other observers. W. E. CARNEGIE DICKSON.

**NEURASTHENIA IN THE ARMY.** (*La neurasthénie dans*

- (24) *l'armée.*) MAURICE BOIGEY, *Rev. Neurol.*, Oct. 30, 1904, p. 1030.

NEURASTHENIA occurs with great frequency in the army, and although the number of cases classed as such in the statistics is small, yet there are a great number of the less severe cases, which are entered under various names, such as "courvature," which are in reality neurasthenia.

In the colonial army about 80 per cent. of those invalided suffer from neurasthenia. This article is chiefly devoted to a study of the condition as it affects the soldier and the officer. In the soldier it is more of the nature of a muscular-physical neurasthenia, while in the officer it is rather cerebral and psychical.

The chief symptoms noticed in the neurasthenia of the soldier are: (1) general muscular asthenia; (2) pains and subjective feelings in the spine; (3) neuralgic pains; (4) internal aches; (5) anæmia; (6) occasionally incontinence of urine; (7) a general tendency to exaggerate the symptoms. Amongst the officers the prominent symptoms are: (1) insomnia; (2) inability to think clearly and difficulty in forming any decision; (3) mental unrest, often passing into melancholy. In addition to these general symptoms there are various special types in which most of the cases could be placed: 1. The dyspeptic. 2. The genital. 3. The hysterical. This last being the least common

of the three, and always characterised by paralysis of an hysterical nature.

In discussing the causation of the condition, chief importance is given to heredity, this being taken in its broadest pathological sense. Amongst other causes, defective education, either excessive or deficient, plays a definite part. All forms of over-indulgence, bodily or psychical, are apt to produce a condition of auto-intoxication, in which the neurasthenic condition is prone to arise.

The article concludes with a brief account of the various methods of treatment which have been found most successful.

T. GRAINGER STEWART.

**A CASE OF EXOPHTHALMIC GOÎTRE WITH MUSCULAR**  
(25) **ATROPHY AND SECRETORY INSUFFICIENCY OF THE**  
**MUCOUS MEMBRANE OF THE STOMACH.** (Ein Fall von  
Basedow'scher Krankheit mit Muskelatrophien und sekreto-  
rischer Insuffizienz der Magenschleimhaut.) ERWIN MIESOWICZ,  
*Wien. klin. Wchnschr.*, Nov. 10, 1904, p. 1206.

THE writer here gives the details of a case of exophthalmic goître with the usual characteristic group of symptoms (*i.e.* exophthalmos, thyroid enlargement, cardiac trouble, tremor and excessive sweating), but in addition to these he describes in this case the presence of symptoms of a somewhat rarer character, viz., lessened resistance of the skin to the passage of the galvanic stream, gastric insufficiency (with excess of mucus and absence of hydrochloric acid both before and after a test meal), and muscular atrophy confined to the upper extremities affecting especially the pectorals and the muscles of shoulder, upper arm and hand. The author refers to the rarity of the last-named condition, and reviews the literature, concluding that in his case, as in the others which he cites, the muscular atrophy is a consequence of changes occurring in the central nervous system, possibly similar in character to those of progressive muscular atrophy, and to some condition of this sort he suggests that the tremor so characteristic of exophthalmic goître is probably due.

W. E. CARNEGIE DICKSON.

**A HITHERTO UNOBSERVED SYMPTOM OF EXOPHTHALMIC**  
(26) **GOÎTRE.** (Ein bisher nicht beachtetes Symptom der Base-  
dow'schen Krankheit.) JELLINEK, *Wien. klin. Wchnschr.*,  
Okt. 1904, p. 1145.

THIS "hitherto unnoticed symptom" of exophthalmic goître is merely the pigmentation of the eyelids which is often observed

early in the course of the disease. This pigmentation may later become less marked. In rare cases it is absent. Jellinek also states that he and Rosin have found that, the number of red blood corpuscles being normal, the iron-content of the blood is diminished relatively more than the hæmoglobin percentage in this disease.

W. E. CARNEGIE DICKSON.

**THE "COMBINED PLANTAR PHENOMENON," A STUDY OF**  
 (27) **THE REFLEXES IN HYSTERIA.** (*Le phénomène plantaire combiné, étude de la réflectivité dans l'hystérie.*) CROCQ,  
*Rev. Neurol.*, Nov. 15, 1904, p. 1069.

THE pharyngeal reflex is frequently lost in hysteria, especially in cases associated with anæsthetic conditions (81%). But this loss is common enough in other diseases. The tendon reflexes are exaggerated (79%), but one can scarcely describe this as pathognomonic of hysteria.

Plantar sensibility is frequently abolished (42%), especially in anæsthetic forms of the disease (63%). The simultaneous disappearance of the ordinary (cortical) plantar reflex, *i.e.* flexion of the big toe, and the medullary plantar reflex, *i.e.* the so-called reflex of the fascia lata (Brissaud) is very commonly found in hysterical conditions (59%), especially in the anæsthetic forms (72%).

Crocq proposes to call this association the "combined plantar reflex," and considers its disappearance of the utmost significance from the diagnostic point of view.

There is no particular relation between this combined reflex and plantar anæsthesia. The deep plantar reflex—muscular contraction in various leg groups, the result of a more vigorous stimulation of the sole—is really a movement of defence, and is often exaggerated in hysteria (50%).

The abdominal reflex varies: usually normal (42%), it may be abolished (24%), or exaggerated (20%).

Ankle clonus (10%), patellar clonus (5%) are described by the author as occasionally found in the disease.

The true Babinski phenomenon he has never found, although in 8% the "fan" sign (abduction of the small toes) was present.

S. A. K. WILSON.



- THE PARADOXICAL FLEXOR REFLEX, ITS RELATION TO**  
 (28) **THE PATELLAR REFLEX AND TO BABINSKI'S PHENOMENON.** (*Réflexe paradoxal des fléchisseurs, les relations avec le réflexe patellaire et le phénomène de Babinski.*)  
 GORDON, *Rev. Neurol.*, Nov. 15, 1904, p. 1083.

SOMETIMES Babinski's phenomenon and other typical indications of a motor path lesion in the cord are absent in cases where one has reason to suspect such a lesion.

Gordon has found a "reflex" in a great number of such cases associated always with exaggeration of the knee-jerk. Seat the patient with his feet on a low stool, turning the limb slightly outwards. Grasp the tibia with the hand and press firmly into the relaxed flexor group of muscles of the calf with the fingers. If the "reflex" is present, there will be *extension* of the big toe or of all the toes. No muscles are involved in the procedure except the flexors, hence the epithet "paradoxical."

In 30 cases of organic disease of the central nervous system involving the medullary motor paths, the phenomenon was elicited, in 12 accompanying Babinski's extensor response, in 9 slightly marked, less distinct than the extensor response, in the rest present in the absence of the plantar extension, or *vice versa*.

In 12 hemiplegics the "reflex" was more easily elicited and distinctly marked on the non-paralysed side.

No hypothesis is proposed to account for the sign.

S. A. K. WILSON.

- ON TACTILE SENSATION.** Sir VICTOR HORSLEY, *Practitioner*,  
 (29) November 1904, p. 581.

THIS address deals, in a somewhat general way, with the representation (*i.e.* the paths and centres) of tactile sensation, as a groundwork of diagnosis of lesions in the central nervous system. The main positions maintained are these:—

(1) Interruption of a peripheral nerve or posterior root causes absolute loss of tactile sensation—the area of anæsthesia is bounded by a sharp line, and there is no trace of overlapping.

(2) There are two types of anæsthesia in lesions of the spinal cord:—

(*a*) "Total cord-anæsthesia," characteristic only of complete division of the cord, *viz.*, absolute anæsthesia up to a certain level. and above this a zone of paræsthesia or hypæsthesia, or, in acute lesions, hyperæsthesia. There is "only one explanation" of this

zone, that it is due to impairment of the grey matter in the neighbourhood of the lesion.

(b) "Root-anæsthesia," in partial lesions of the cord. Head's areas are absolutely correct, they do not overlap, the anæsthesia is absolute.

(3) Personal observations "absolutely confirm" the view that the fillet-fibres terminate in the optic thalamus, none going straight through to the cortex. Injury of the fillet-region causes anæsthesia of the cord type, *i.e.* absolute.

(4) From the optic thalamus, a new set of fibres conduct sensory impulses to the cortex. Experimental lesions of the thalamus show that these fibres can be traced into the Rolandic area as well as towards the middle aspect of the hemisphere. The gyrus fornicatus is one centre for tactile impressions, but sensation is represented in more than one part of the cortex. The writer believes that he has unimpeachable clinical evidence that tactile sensation is represented in the so-called motor centres, lesions of which cause not only localised paralysis, but also localised sensory loss, and also that there is a quantitative relationship—the extent of anæsthesia being in proportion to the amount of cortex involved: the anæsthesia is not absolute, there is slight loss of actual sensation of touch, and there is also loss of the more important ability to localise exactly the point touched. These characters of the sensory loss point to a lesion somewhere in the motor region of the cortex, and are not found in deeper lesions below the optic thalamus. The kind of anæsthesia has, therefore, great value as a means of differentiating seats of lesion.

A. W. MACKINTOSH.

#### NOTES ON CASES OF PULSATING EXOPHTHALMOS. C. H.

(30) USHER, *Ophthalm. Review*, Nov. 1904, p. 315.

CASE 1. *Right traumatic pulsating exophthalmos; ligation of right external and internal carotid arteries; death; post-mortem examination.*

Right eye: no perception of light; pupil half dilated, no action to light; proptosis marked; movement of globe nearly abolished in all directions; pulsation felt when fingers are laid on the lids. The patient died five days after the injury. On post-mortem examination a large rupture of the internal carotid artery into the cavernous sinus was found, but it is difficult to account for its presence. There was no fracture of bone causing injury to the vessel, no evidence of a pellet having entered the orbit or cranium (he had been shot), and no disease in the artery, such as atheroma.

or aneurysm. A prominent feature in this case was marked visible pulsation of the neck on the side of the lesion.

CASE 2. *Left spontaneous pulsating exophthalmos; ligation of left internal and external carotid arteries; recovery.*

The arteries were ligatured in 1898, two months after the onset of the symptoms. The patient has been working constantly as shepherd since he left the Infirmary shortly after the operation, nearly seven years ago. Vision of left eye is  $\frac{3}{4}$  partly. At the present time no thrill is felt or bruit heard over the globe. There is evidence of marked arterio-sclerosis in many of his blood-vessels. There is reason to believe that the cause of the symptoms in this case was an aneurysm of the internal carotid or of the ophthalmic artery.

CASE 3. *Left traumatic pulsating exophthalmos; ligation of left common carotid; in a child four years of age.*

The artery was ligatured about ten weeks after the injury. Eighteen months later, proptosis was still present, but no pulsation of globe and no bruit; the pupil was wide and did not contract to light or on accommodation; movements of the globe were full.

AUTHOR'S ABSTRACT.

#### A PREGNANCY TOXÆMIA OF THE CENTRAL NERVOUS (31) SYSTEM. (Graviditätstoxonose des Centralnervensystems.)

L. BRAUER, *Münch. med. Wchnschr.*, No. 26, 1904.

Two years ago the author described a case of a woman who with each pregnancy developed hæmoglobinuria, icterus, and general nervous symptoms during the last month of gestation. This form of illness was confirmed by Meinhold, and also by Schmorl. Shortly after his first observation, Brauer came across a case of recurring icterus in pregnancy, without simultaneous hæmoglobinuria, which was fully described in Hegar's *Beiträge*. He thought it possible to bring these symptoms into definite etiological relationship with the processes of metabolism during pregnancy, and thus range them in one large group along with pregnancy kidney, eclampsia, and acute yellow atrophy of the liver. With the conception of a placental toxin and syncytial action, one can now connect the so-called auto-intoxications of pregnancy with the metabolic relations which exist between the maternal and foetal organism. "Foetal cells and products of metabolism exercise a poisoning influence on the maternal organism" (Veit).

To this chain Brauer now adds a further link, and this is a disease of the central nervous system dependent on the processes of pregnancy—an example of the effects of a pregnancy toxæmia.

The patient had had sixteen labours which yielded ten living children. When five months advanced in her fifteenth pregnancy she complained of rheumatic pains in both limbs, and formication. There was no "girdle" sensation. Soon afterwards the right arm and leg became paretic, and in the last month of pregnancy the paralysis involved the left side also. The labour was normal, and was followed by speedy improvement. In a month the patient could walk about, and two months later was quite well.

About two years later the next confinement took place. During the first and second months of this pregnancy there was an uncomfortable sensation in the feet, and headaches were also complained of. Between the fifth and sixth months there was marked dragging of the right foot, and soon the woman could only walk a few steps. There was formication confined to the right side. Up to nearly full time the paralysis steadily increased, and both sides of the body were then completely paralysed. Dyspnoea was present in the last weeks, there was partial aphonia, but no speech or eye disturbances were noted. Insomnia was marked. Accompanying the paralysis, cramp-like pains—at first slight, and later on very severe—were complained of. As labour was nearing term, dropsy of the legs and arms appeared, but the face was not affected. There was no albumen in the urine.

One month after labour the patient could move her head and legs, and the formication was gone. Examination showed that she was anæmic. The cranial nerves were normal; no nystagmus or ocular paralysis. The right arm was completely paralysed; the muscles were tonically contracted; and anæsthesia was also present. Although the patient was emaciated, there was no atrophy of the muscles. Tendon and deep reflexes were increased. Babinski's sign was present on both sides.

The case is of interest, both on account of the peculiar etiology, and also for the mixed nerve symptoms present. There was undoubtedly a lesion in the upper cervical cord, and that lesion was probably a myelitis in the wider sense of the word. Hysteria can be excluded. The complete cure of the first mild attack, the complete cure also of the second more severe attack, and the slowly progressive symptoms in each case, all go against the theory of a simple inflammatory myelitis with destruction of tissue. There is much that points to the case being one of multiple sclerosis—atypical and essentially spinal—for it is known that this disease can account for the most diverse symptoms, and in its origin and progress is not seldom found in dependence on pregnancy. The absence of nystagmus, scanning speech, and intention tremor go against this diagnosis. Brauer prefers to regard the case as a "toxic myelitis" in which there was a degeneration of the nerve elements, but no inflammatory process.

Other pregnancy toxæmias have been described in literature, but it still remains to make clear what processes are responsible for the onset of such conditions during this period. Recent work on eclampsia, pregnancy, kidney, and hyperemesis gravidarum has thrown some suggestive light on the subject.

OLIPHANT NICHOLSON.

## PSYCHIATRY.

**THE PSYCHOLOGY OF HALLUCINATION.** W. H. B. STODDART,  
(32) *Journ. Ment. Sci.*, Oct. 1904, p. 633.

THE paper is an attempt to demonstrate that hallucination is a disturbance of ideation rather than of perception or simple sensation, and that its physical basis is therefore identical with that of ideation, being situated in the association-areas of the cortex.

After demonstrating, by means of certain elementary stereoscopic and other figures, that there is a tendency to place ideational content in sensory experience, the paper goes on to show that the hallucination process consists of a positive and a negative side, the positive side being an increased associative activity and the negative side being diminution of sensation in the particular sense-department affected. Patients suffering from hallucinations of vision do not see objects in the neighbourhood of the hallucination-image, and those with hallucinations of hearing are, for the moment, partly deaf. This negative factor is, however, occasionally supplied by artificial means, the patient closing his eyes or covering his ears with his hands.

Another factor of hallucination is diminution of sensation in other sense-departments than that affected, and instances are given of auditory stimuli being inhibited by visual stimuli.

Special reference is made to the epigastric and allied sensations, such as globus hystericus, neurotic spine, hysterical hip and hysterical shoulder. It is contended that these symptoms are associated with anæsthesia of the type described by the author in 1899. The distribution of this anæsthesia is such that the abdominal and neighbouring areas remain, for the most part, unaffected, and charts illustrating this distribution are given in the paper.

Consciousness, being entirely dependent upon sensation, is in such patients entirely dependent upon sensations derived from the abdomen or neighbouring parts. These accordingly demand a large amount of the patient's attention, and become the seat of abnormal sensations.

Special reference is made to the epileptic aura, to which the above principles are applied.

AUTHOR'S ABSTRACT.

**HALLUCINATIONS.** By W. A. WHITE (Washington), *Journ. of*  
(33) *Nerv. and Ment. Dis.*, Nov. 1904, p. 707.

WHITE insists on a painstaking examination of the organs of the special senses in all cases with hallucinations. He claims to have found in all such cases in his experience a peripheral pathological process which could explain the hallucinatory phenomena. Hallucinations are false perceptions, and the mental state in illusions and hallucinations is the same—a central derangement causing a false perception of sensory elements. He quotes ten personally observed cases to support his conclusions.

C. MACFIE CAMPBELL.

### TREATMENT.

**THREE CASES OF EXOPHTHALMIC GOITRE TREATED BY**  
(34) **THE BLOOD AND BY THE SERUM OF THYROIDLESS**  
**SHEEP.** (Trois cas de goitre exophtalmiques traité par le  
sang et le serum de moutons éthyroïdes.) P. SAINTON and  
B. PISANTE, *Rev. Neurol.*, Nov. 30, 1904, p. 1109.

THE writers first refer to the work already done in relation to the serum treatment of exophthalmic goitre. Thus, Ballet and Enriquez were the first to carry out the treatment by injections of the serum of thyroidless animals. Möbius, in Germany, published in 1901, "Observations on Three Cases of Exophthalmic Goitre" which had improved on injections of the serum of a thyroidless sheep. This serum has since been prepared by Merck under the name of "antithyroidine." Several cases have been treated by the serum administered orally and by injections. Hallion employed the blood instead of the serum. Jean Lépine has recently obtained an antithyroid serum from animals immunised against hyperthyroidism. The authors, however, make no reference to the reviewer's observations on the action of a similar serum prepared by himself, nor to the results obtained by Lanz and others by employing the milk of thyroidless goats in the treatment of exophthalmic goitre. Three cases are detailed by the author which were treated by the serum or blood of thyroidless sheep. One case was considered to be cured by the treatment, and the others improved. Attention is specially drawn to the favourable action of the treatment on the frequency of the pulse as evidence of its efficiency. Preference is given to the oral administration of the serum, which proved to be more active than the glycerinated blood itself. All three cases had resisted other medical treatment, the first having been treated previously by thymus extract, the second by electricity, and the third had been

stationary for three years ; further, the authors have not obtained such favourable results in cases of exophthalmic goitre by any other means of treatment.

GEORGE MURRAY.

**CONTRIBUTION TO THE TREATMENT OF EPILEPSY BY**

(35) **CENI'S METHOD.** (*Contributo alla cura dell' epilessia col metodo Ceni.*) GIOVANNI TIENGO, *Riv. Speriment. di Freniat.*, 1904, Vol. xxx., f. ii.-iii., p. 520.

THREE years ago Ceni communicated the results obtained by him in the treatment of epilepsy by serotherapy. After some preliminary and ineffective experiments, which, however, showed the presence of a special toxin in the blood of epileptics, he undertook two series of experiments. In the first, he injected epileptics with blood serum derived from other epileptics. In the second, he practised auto-serotherapy, re-injecting into patients the serum of blood which had been withdrawn from them previously. From these experiments he concluded that there are two active principles in the blood of epileptics, with different and antagonistic properties. One circulates in a free state in the blood and possesses purely toxic properties: the other is found in the blood only in a latent state, and is endowed with stimulating qualities for the cellular elements in which metabolism takes place, and which constitute also the probable site of the elaboration of epileptogenous toxic agents.

The action of these principles sometimes has a beneficial character, sometimes a harmful, according to the special organic conditions of the individual in whom the serum is injected.

Ceni's method of treating epilepsy by the injection of serum has been used by various observers, but with widely differing results. Tiengo gives a detailed account of six cases treated by himself and sums up as follows :—

The patients were all the subjects of grave epilepsy, either from the frequency, duration, and intensity of the motor disturbances, or from the long existence of psychic phenomena. They had all been on bromide treatment, but on beginning treatment by Ceni's method, the bromide was reduced either suddenly or gradually. Of the six patients under treatment, five showed a notable improvement, while one re-acted so as to contra-indicate further experiment. The general bodily conditions were much improved, the gain in weight being a prominent feature.

From an average of fifty fits in the month in one patient, the number fell in the first month of treatment to twelve, and subsequently to three or four. In another case, in which the disease had lasted for about twenty years, and there were on an average three or four fits a week, even better results were obtained.

T. C. MACKENZIE.

## Review

**PHYSIOLOGICAL AND CLINICAL INVESTIGATIONS ON THE BRAIN.** (*Physiologische und Klinische Untersuchungen über das Gehirn. Gesammelte Abhandlungen.*) EDWARD HITZIG, Berlin, 1904. 27 M.

UNVANQUISHED by his enemies, but conquered by the overpowering destiny which robbed him almost completely of the sense of sight, arrested prematurely in the pursuit of his work, Hitzig gives to the scientific world his treatises on the brain.

Hitzig is the creator of modern brain physiology. A period of more than thirty years, full of success, but full also of conflict, was occupied in this pursuit. He was attacked not by honourable scientific methods only; he was often misinterpreted, deprived of his intellectual discoveries, or even ignored. But equipped as a master for the scientific warfare, a sharp opponent, a severe judge, a stern critic of himself, accurate to a degree hitherto unknown both in his experiments and in the topical deductions derived therefrom, he came forth from the strife of pens a victor, with fresh facts and ever new points of view as his reward.

Hitzig, as is well known, is the founder of the doctrine of brain localisation, and the history of the origin and development of this theory forms the contents of the first part of his book. Here we find, as it were, the preface and the centre of the whole in that famous discovery which he made in 1870, in conjunction with Fritsch, of the electrical excitability of the cortex—a discovery which has permanently established that doctrine of Flourens of the equivalent value of each part of the cortex, which has become the turning-point of all our knowledge of the physiology of the brain. And in what wonderful continuity are all Hitzig's subsequent investigations linked to these main, ingenious electrical experiments: the production of paralysis by brain extirpation; the observation and explanation of cortical epilepsy; the discovery by the process of comparative anatomy of the muscle centres of the ape; the first investigations into circumscribed focal diseases of the human cortex.

We know that H. Jackson had already supposed a localisation in the cortex, and that Ferrier, Luciani, and many others published similar investigations later. But the first real discovery of the electrical excitability of certain parts of the cortex (the motor region) and, what is perhaps almost as important, the first complete and profound investigation of that region, with an immense number of experiments, and the keen conclusions which he derived from them, leading up to the modern result of surgical brain diagnosis—



all this will be to Hitzig's everlasting credit, though, apparently from ignorance of much of his work, it may be attributed to others, even by his own countrymen. Nevertheless, many of his opponents became his friends, and honour and recognition from the most eminent of his fellow-workers (Charcot, Jackson) have not failed him.

The cases of brain surgery given in the first volume afford good insight into this theory as it at present stands; and it contains further, an inexhaustible wealth of important facts and ideas which, although written more than thirty years ago, appear so fresh and modern, that one wonders why they have not already been appropriated by every neurologist, not to say by every physiologist and psychologist. Among these essays I would specially mention those on the derangement of the muscular innervation and of perception of position in space caused by galvanisation of the head (vertigo, movements of the eyeballs, which, although wrongly interpreted by Hitzig, led him to his first famous discovery): on the cerebellum; on certain anomalies of muscular innervation; on hemiplegic contracture; on corresponding movements.

The second part of the work is also based on localisation. It refers exclusively to the brain of the dog. These essays were published first in the *Arch. f. Psychiat. und Nervenheilk.*, 1901-1903. Hitzig had formerly described the derangement of movement in the limbs of the dog caused by injury to the gyrus sigmoides as a "defect of will-power." Now we know that the same disturbances may also occur when the occipital portion—the "seeing sphere"—of the brain is injured, but in any case they do not arise directly from injury to the cortex, but indirectly, since it is impossible to restrict the lesion exclusively to the cortex.

Another comprehensive chapter deals with the onset, symptoms and course of disturbances of vision after operations in the motor zones, with a scrutiny of Munck's theory as to the function of the occipital brain, and the significance of those experimental results as applied to our knowledge of brain mechanics. These investigations were undertaken on account of the work of his opponents, of Munck especially. That Hitzig did succeed in overcoming his adversaries in this region is due, firstly, to the fact that no one has made so many experiments in this direction as he; and secondly, to the methods which he discovered, such, for instance, as that of comparing the field of vision of a man with that of a dog which has been suspended so that all its limbs are free to move, and then graphically recording the result; and lastly, to the clearness and conciseness with which he set forth his questions and fearlessly laid bare the blanks in our knowledge of the sensori-motor and visual functions.

It is impossible here to go into the wealth of new facts and theories which are set forth in this work regarding the relations of the motor and sensory functions to the various parts of the brain: restitution and inhibition; the function of the infra-cortical centres; our knowledge of optical perception; therefore we must limit ourselves to some special points. Projection in Munck's sense of the word, viz., that certain territories or elements of the retina act only in conjunction with certain corresponding territories of the cortex, does not exist. Cortical blindness (that is, total permanent blindness resulting from injury to the occipital cortex) does not exist. Such a disturbance of vision must be regarded as essentially contralateral homonymous hemianopia. The site A1 in Munck's sphere of vision has no close relation to the macula lutea. Munck's "soul-blindness" in the dog is nothing more than amblyopia. A great part of the seeing process in the dog takes place in the subcortical ganglia. The functions of these are affected by lesions either in the anterior or the posterior portions of the cortex, but they soon recover, and even acquire a certain independence, so that in future lesions of the cortex do not necessarily produce visual disturbances.

Hitzig's idea of the mechanism of visual perception may be shortly given in his own words. "For me, commencement of all vision consists in the production of the perfect optical image in the retina; continuation of vision in the combination of this optical image with motor, and perhaps with other sensations of innervation, giving rise to representation of a lower order in the infra-cortical centres; and the highest development of vision—dependent on the existence of the cortex—consists in the perception of these lower representations and their association with ideas and sensations of different origin."

What is the reason that the electrical excitability of the brain was not discovered before Hitzig? Investigators had always hitherto operated on the posterior part of the brain, where no motor muscular centres lie, and where operation is easier; he operated on the frontal region, and thus made his discovery. This seems a very simple matter in the retrospect; so does the story of Columbus's egg.

M. RHEIMBOLDT, Hissingen.

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# Review of Neurology and Psychiatry

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## Original Articles

### **THE ENDOCELLULAR FIBRILLARY RETICULUM AND ITS RELATIONS WITH THE FIBRILS OF THE AXIS-CYLINDER.**

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IN the *Rivista sperimentale di Freniatria* (vol. xxx., fasc. ii.-iii., 1904), and in the *Annali di Neurologia* (fasc. i.-ii., 1904), I have published the methods which I have used for some years for the coloration of the endocellular fibrillary reticulum, the presence of which I succeeded in demonstrating, in 1896, in the protoplasm of the nerve cells of vertebrates (1). These later methods are modifications of the methods which I used in 1896 with which, besides staining the endocellular reticulum, I was able to stain the pericellular or peripheral reticulum.

The modification applied to this first method has consisted in attempting to obtain results from tissues which had been previously fixed and hardened; these results have been obtained by making use of the peculiar properties of "pyridine."

By means of the new methods, I have been able to definitely demonstrate the existence of the endocellular fibrillary reticulum, which I have described, and also the long fibrils, described by Bethe (2). The interest of the demonstration of the existence of the fibrillary reticulum and of the long fibrils, of which I published a note in 1900 (3), is obvious.

But it was the supposed existence of the long fibrils only which Bethe regarded as the morphological basis of his theory

with regard to the course of the nervous stimuli, a theory, according to which he considers the nerve cell in vertebrates to be a simple zone of passage of nervous stimuli, while the true nervous function takes place in the spaces between cell and cell, *i.e.* in a hypothetical interstitial network.

Now, the morphological basis of Bethe fails to explain the relation of the close and rich endocellular fibrillary reticulum to the fibrils of the protoplasmic processes and to those of the axis-cylinder prolongation.

The evident richness of the reticulum of anastomotic fibrils has been admitted by all who have seen my preparations. At the International Congress of Physiology at Turin (1901) I again referred to my results, and showed microscopical specimens.

I compared the demonstration of the endocellular reticulum with the findings of Bethe, and I suggested that its relation with the fibrils of the protoplasmic processes and of the axis-cylinder indicated the functional importance of the endocellular reticulum, and at the same time the insufficiency of Bethe's theory.

As a hypothesis, I offered the opinion that the endocellular reticulum might represent an apparatus for the reception and synthesis of the nervous stimuli (4). I also made a similar communication, with a demonstration of specimens, to the 11th Congress of the "Societa Frenetica" of Italy in 1901 (5).

I thus demonstrated the existence of two fibrillary systems in the nerve cells of the cerebro-spinal axis: the first lying in the cell body and forming by an anastomosis of fibrils a fine reticulum, which is in close connection with the fibrils of the protoplasmic processes and of the axis-cylinder; the second simply passing through the nerve element.

Later, I called attention to cells in which the two systems are not present, but only one, which is supplied by the endocellular fibrillary network. These elements, which are found in various parts of the nerve centres, are most evident in cells which possess few prolongations: for example, I have met with and described this type of cell, provided with only one system, scattered amongst other cells presenting the two systems, in the anterior portion of the ventral nucleus of the acoustic nerve (6). But even in these elements the relation of continuity between the reticulum and the axis-cylinder is evident, as may be seen in one of the figures of that publication.

In 1902 I referred to the endocellular reticulum, and gave a demonstration of specimens, at the first Congress of Pathology held at Turin (7), and I called attention to some conditions which were present frequently or almost constantly throughout the course of an extensive research. One of these was a characteristic condensation of the reticulum towards the centre of the cell, *i.e.* in the perinuclear portion. To this condensation, which is more marked in cells with many prolongations, I gave the name "perinuclear circle or ring," "*cercine o anello perinucleare.*"

In well differentiated preparations there was a constant absence of coloration of the nucleus; but when the differentiation was not complete, the nucleus was slightly stained, and then between the nucleus and the surrounding margin of the fibrillary reticulum and of the perinuclear ring there remained a clear zone; but when the differentiation was complete, the nucleus appeared as a white mass, without any obvious structure.

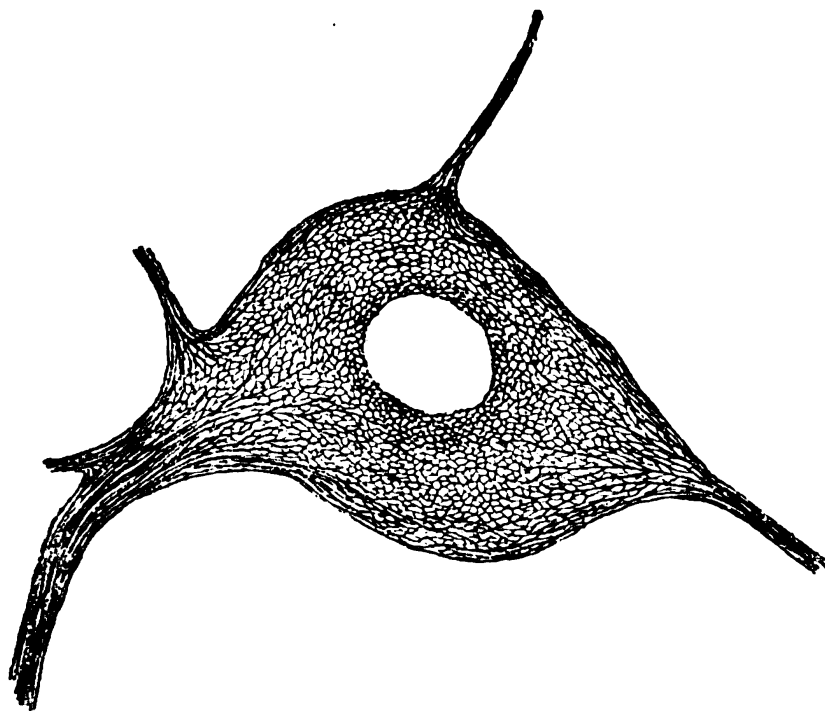


FIG. 1. (This figure was referred to in the author's communication to the International Congress of Physiology in Turin (1901), and has been published in the first edition of the "Treatise on Physiology," by Luciani, Vol. ii., p. 220, *Società editrice libraria*, Milano, 1902.)

These characteristics are represented in the accompanying figure, which I showed in my communication to the International

Congress of Physiology at Turin (1901), a figure which exhibits a cell from the anterior cornu of the cord of a dog.

They may also be seen in the figures illustrating the cellular elements of the acoustic nerve in the above mentioned article.

I also offered the opinion at this Congress that "the theory of Bethe on the functional importance of the cell, and on the course of the stimuli, was evidently insufficient." I made an identical statement at the International Congress at Madrid in 1903, and I also showed microscopical specimens there.

By means of my selective methods, it is possible to stain the endocellular fibrillary reticulum or the pericellular reticulum at will, and, perhaps, it will be well if I here offer a few remarks on the pericellular reticulum. The methods, which have been rendered selective by the employment of pyridine, demonstrate a pericellular reticulum similar to that which I observed in 1896, *i.e.* an investment having a reticular character. This was seen in 1893 by Golgi, and more fully described in 1898. It corresponds also to the pericellular reticulum, which, in numerous publications, has been described by Bethe, Semi Meyer, Cajal, Held, Turner, Simarro, Cavalié, and other authors; it is not analogous to that described by Auerbach and by Shinkishi Hatai. The principal results, in relation to the pericellular reticulum, which have been obtained by the old method and by the successive modifications, are the following:—

(a) A demonstration of anastomoses between the peripheral reticulum and the surrounding tissues. I was first able to show this in 1896, and I have confirmed it since. Bethe claims to have demonstrated that nerve fibrils unite in the peripheral reticulum (8). From a careful examination of my preparations, I have come to the conclusion that a large proportion of the fibrils of the surrounding tissue, which have anastomosed with the pericellular reticulum, must be of neuroglial nature (9).

Hans Held has recently admitted that he was mistaken in his opinion, which is analogous to that of Semi Meyer, that the threads of the pericellular reticulum were nerve ramifications, and he now admits their neuroglial connections (10).

(b) A demonstration of the existence, in the middle of each mesh of the pericellular reticulum, of an apparatus which consists of very fine fibrils arranged in a network, or radiating from a central point. To this system of fibrils radiating from the

central point I have applied the name "radiations" ("raggiere"). The existence of this structural element has been confirmed by Held, who admits that the "raggiere" correspond to the appearances which he described under the name "sternförmigen Haufen." But while I did not express any opinion as to the value of the "raggiere" in 1901, Held said that he considered they were of nervous origin. Cajal, however, has stated that, in all probability, the central point of these formations corresponds to the terminal buttons of Auerbach, which he has demonstrated by his method. The "raggiere" possess a characteristic structure which the method of Cajal does not stain. But whatever may be their significance, there can be no doubt about their existence.

Confirmation of the existence of the endocellular fibrillary reticulum, by means of a selective method, has been provided by Ramon y Cajal during the past year. Cajal has described his method in the *Archives latines de Biologie*. According to him, "las células de mediana talla de la médula, cerebro, talamo, etc., presentan las neurofibrillas al parecer anastomosadas engendrando la red de que habla Donaggio" (11).

Cajal's method is based on the reduction of silver, as are also the methods of Bielschowsky (12), and of Robertson (13), who had made some researches in this direction before 1898, and who has just recently published the technique of his method. But the method of Cajal demonstrates the endocellular reticulum, which I have described, only partially; in fact, as I have shown clearly in my successive publications, the metallic impregnation of the motor cells of the spinal cord, or of the large pyramidal cells of the cerebral cortex in adult animals, does not bring out the reticulum at all clearly (14).

Van Gehuchten also has found that, in the cells of the cortex, in the cells of the spinal cord, and of the reticular formation of the bulb and pons varolii, the method of Cajal leaves the existence of the reticulum in doubt.

Now, all this uncertainty is due to the imperfection of Cajal's method. This method has not yet succeeded in demonstrating the endocellular reticulum with that certainty which for years I have obtained in the case of the cells of the whole cerebro-spinal axis.

The demonstration of the endocellular fibrillary reticulum has led Cajal to adopt the same fundamental opinion which I have formed from the examination of my preparations, and which I expressed at the International Congress of Physiology at Turin, and which I repeated at the first Congress of Pathologists in Italy in 1902, *i.e.* that the reticulum represents a condition which offers the strongest opposition to the idea that the cell is simply a zone of passage of independent fibrils, put forward by Albrecht Bethe, and adopted by him as the basis of his theory on the course of the stimuli.

Cajal holds that all the fibrils anastomose, although his method is so little adapted to support such a statement, in so far that it leaves one in doubt even of the existence of the reticulum in cells, in which, with my methods, it is perfectly obvious.

Cells do certainly exist in which all the fibrils anastomose, as I have shown in some of the cells of the acoustic centre; and other elements of the cerebro-spinal axis seem also to be provided with an endocellular reticulum only, which is connected with the fibrils of the protoplasmic processes. In any case, I have been able to demonstrate by my researches that there are at least two cellular types:—

1. Elements provided with an endocellular reticulum only. (See the above-mentioned publication on the acoustic centres.)

2. Elements provided with two systems of conduction, *viz.*:  
(*a*) consisting of fibrils which form the endocellular reticulum, and (*b*) consisting of fibrils which pass through the cell body and still preserve their individuality. These, which constitute the majority of nerve cells, I described first in 1900, and I demonstrated them also at the International Congress at Turin in 1901. Both of these types of cells exhibit a condition which is strongly opposed to the theory of Bethe, *viz.*, the endocellular reticulum which I have described. In my opinion, also, the application of the method of Cajal may lead to erroneous conclusions in the field of pathological histology. In fact, researches which I have made with Fragnito on the lesions of the endocellular fibrillary reticulum in the cells of the cord after tearing out the sciatic nerve with its corresponding roots in the rabbit, researches carried out with my method and controlled by Cajal's method, prove that Cajal's method would lead one to believe that the fibrils were either destroyed or

diminished in cells in which, by my method, they were evidently well preserved.

On this account, also, the results obtained by Marinesco with Cajal's method in pathological material must be controlled before they can be accepted. My methods have been described in several publications (15), and may be classified as follows:—

(a) Coloration of the endocellular fibrillary reticulum and of the long fibrils (met. 1-2 for the coloration *in toto*, and met. 3-4 for the coloration in sections).

(b) Coloration of the endocellular fibrillary reticulum, of the long fibrils, and of the granules (met. 5).

(c) Coloration of the pericellular reticulum (met. 6-7).

(d) Simultaneous coloration of the pericellular reticulum, of the endocellular fibrillary reticulum, and of the long fibrils met. 8).

(e) Coloration of the axis-cylinder of the nerve cell.

*Note.*—An account of methods 3, 4 and 7 appeared in the *Review of Neurol. and Psychiat.* in September 1904, in Dr Ford Robertson's abstract of a communication which I published in the *Annali di Nevrol.*, Anno xxii., fasc. 1-2. I wish here to give a short note on method 5, which has yielded good results, and also on methods 1 and 2.

#### *Method 1.*

The tissues are passed through the following stages:—

1. Pieces of tissue, 2 or 3 mm. thick, are fixed in Heidenhain's sublimate solution for 24 hours. Excess of the sublimate is removed with iodine solution.

2. After washing in distilled water for 2-3 hours, they are placed in pyridine for 48 hours, changing the pyridine after 24 hours.

3. Stain the pieces—attached by one border with paraffin to a bit of cork and suspended in the colouring fluid—in an aqueous solution of thionin, 1-10,000 or 15,000, for 48 hours, renewing the stain after 24 hours.

4. After immersion of the pieces in aqueous solution of molybdate, 4 per cent., to which has been added hydrochloric acid in the proportion of 1 minim to 1 gramme of molybdate, wash in water, renewed often, for 12 hours, and embed in paraffin.



*Method 2.*

proceeds as in method 1, but after the pieces have passed through pyridine, wash in distilled water for 24 hours, renewing it several times; then immerse them in the solution of molybdate as before, and after washing for 5-10 minutes, pass them through stages 3-4 of method 1.

*Method 5.*

Pass the pieces through stages 1-2 of method 1; then wash in distilled water for 24 hours; after a few hours the tissue should be cut into pieces not more than 2-3 mm. in thickness. The water must be changed frequently, and the last washing should be done in a fresh vessel.

Next, place the pieces in an aqueous solution of molybdate of ammonia, to which has been added some hydrochloric acid—1 minim to 1 gramme of the molybdate—for 24 hours. The solution of the molybdate should be fresh, and the pieces should be kept on their edge. Now wash in water, changing it a few times, for 24 hours, and proceed to embed in paraffin.

When cut, place the sections on coverslips and dry. Remove the paraffin in the usual way, and stain with an aqueous solution of thionin (1-10,000). The solution of thionin should not be too old.

The succeeding procedure may be one or other of two:

(a) Pass the sections successively through distilled water, ordinary alcohol, absolute alcohol and xylol, and then mount in Grüber's neutral balsam.

(b) Distilled water, ordinary alcohol (the alcohol must be thoroughly washed out), the ammonium molybdate solution for 15-30 minutes, distilled water for 15-20 minutes (renew the water a few times), ordinary alcohol, absolute alcohol, xylol, neutral balsam.

Method 5 is thus a combination of parts of methods 1 and 3. For a more detailed description of these I must refer the reader to Dr Robertson's abstract.

I will now proceed to discuss the relations between the endo-cellular fibrillary reticulum and the fibrils of the axis-cylinder.

By my previous researches, I have established the fact that the axis-cylinders of the nerve cells of vertebrates receive fibrils

from the endocellular reticulum, which I have described and illustrated in several previous communications. The axis-cylinder may derive fibrils from the fibrillary reticulum alone, or from the fibrillary reticulum and from the long fibrils at the same time, when the cell contains the two fibrillary systems.

The two systems contribute more or less to the formation of the axis-cylinder; but it is certain that, excepting in special instances, the number of long fibrils which pass from the cell body directly to the axis-cylinder is small. While, however, the axis-cylinder derives few fibrils from the long fibrils, it obtains a large number from the endocellular fibrillary reticulum.

But it must not be supposed that the origin of the axis-cylinder from the reticulum takes place according to a uniform scheme; nothing could be more varied or more complex than its origin. It would be possible, I think, to establish, from this point of view, a series of forms, some of which would be considered as characteristic. These appearances would depend on the type of the endocellular reticulum, the various conditions of the condensation, and the diverse positions of the long fibrils.

The type which is least complex in the relations between the reticulum and the axis-cylinder prolongation, is seen among the cells of the anterior portion of the ventral nucleus of the acoustic nerve. In this region, side by side with the cells which possess both the endocellular reticulum and the long fibrils, are found cells which exhibit only the system derived from the endocellular fibrillary reticulum.

Some of these cells, as I have stated before (16), have an irregular shape, but their fibrillary reticulum presents itself in quite a regular form, which may be round or oval.

The fibrillary reticulum, which in these cells is deeply seated, has nothing to do with a coarse reticulated structure, which can be seen in the same cells. This I have described in another publication thus: "If, instead of differentiating so as to produce the selective coloration, I leave the coloration diffuse, the border of the cell exhibits a coarse reticular structure, which is continued towards the centre of the cell, intersecting the fine, deeply-coloured threads of the fibrillary network.

This reticulated network is met with in all nerve cells; it is not stained by my methods excepting with special technique, and even then it assumes only a pale coloration, and it often has the

aspect of intersecting trabeculae. The nucleus is often stained at the same time.

In all probability this network corresponds to the trabeculae which sometimes appear when using Nissl's method.

To these data, which I consider interesting from several points of view, and to the technique which is best suited to bring them into evidence, I shall draw attention in another publication. Here I wish to add that there is nothing in common between the true fibrillary reticulum, which I have described, and the trabecular network (spongioplasm?), which I have just mentioned.

In the cells of the acoustic nucleus, of which I have spoken, the fibrillary reticulum is quite simple. It does not give rise to that perinuclear fibrillary condensation which I have termed the perinuclear ring, and the origin of the axis-cylinder, which springs from the reticulum at one pole of the cell, shows no special peculiarity.

As a rule, I find that it is the medium and small cells which most frequently belong to the type of cell which is furnished simply with an endocellular reticulum; in the cerebral cortex this type is found especially in the layer of polymorphous cells. Some of these, although provided with a certain number of protoplasmic processes, present a complete reticulum, the meshes of which are often narrow and close, so that they colour more darkly than those of the surrounding cells. In these the axis-cylinder springs from one end of the cell without any special fibrillary condensation.

A similar arrangement is found in other parts of the cerebrospinal axis. In the smallest cells also we meet with the prevalence, or even the exclusive presence, of the endocellular reticulum, which, in spite of the small size of the cells, is plainly demonstrated by my methods.

Fig. 12, pl. x., represents one of the smallest cells of the posterior horn of a rabbit, provided with the reticulum in connection with a protoplasmic process, and with a slight suggestion of a perinuclear ring.

Analogous types are represented in figs. 13-14, pl. x.; 10-11, pl. ix.; 7-8, pl. viii. (cells of medium size).

But even the medium and small cells, like the large elements, may present both the fibrillary reticulum and the long fibrils. These long fibrils may be confined to an extremely narrow zone

at the periphery (fig. 15, pl. x.), or they may be more abundant and arranged either at the periphery or at the centre of the cell, or both at the same time.

Now, since the mode of origin of the axis-cylinder often depends on its relation with the structure of the perinuclear ring, as well as on the type of cell, I think it is necessary to say something about the morphology of this ring. The perinuclear ring, to which I drew attention some time ago (17), is produced by a condensation of the fibrillary reticulum around the nucleus. The fibrillary reticulum, it is true, is scattered throughout the cell, but, very frequently, the further it is followed towards the centre of the cell, the more it is found to be crowded together.

The method of Bethe, on the other hand, shows either an absence or a great rarefaction of the fibrils in this region.

The condensation appears often to depend on the fact that in the most central part of the cell, groups of fibrils undergo a subdivision (fig. 1 in the text), but it is also observed in other conditions in which the meshes of the network become smaller and more crowded together around the unstained nucleus. This perinuclear ring must not be confused with those bands which are derived from the passage of the long fibrils through the centre of the cell and form a sort of cap to the nucleus (*cuffia perinucleare*).

Therefore, while one can say that the fibrils of prolongation *b* of fig. 11, pl. ix., having entered the cell and spread out in the shape of a fan, contribute to the formation of the endocellular reticulum and also of the perinuclear ring, yet it can be easily seen that not all the fibrils of the prolongations *a*, *b*, *d* of fig. 9, pl. ix., participate in the formation of the condensation, but a considerable number sink deeply into the cell and form a perinuclear cap as they pass on to form the axis-cylinder.

These perinuclear caps are found more or less frequently in the cord, in the bulb, and in the brain, in the large and medium and sometimes in the small cells, and I repeat that they consist of fibrils passing through the cell, although they are associated with fibrils which participate in the formation of the perinuclear ring. There may be present around the nucleus only the perinuclear ring, or there may be this and a perinuclear cap together (fig. 9, pl. ix.). The perinuclear ring is sometimes connected with other zones of the endocellular fibrillary reticulum which

are equally compressed (fig. 2, pl. v.), and which are often situated towards the periphery of the cell (fig. 7, pl. viii.; figs. 10-11, pl. ix.; fig. 16, pl. x.). These forms are frequent in the rabbit, but much less frequent in the dog, the cat, and in man.

In the dog the reticulum is more uniform and regular (fig. 1 in the text, and fig. 16, pl. x.). Moreover, by the confluence of the fibrils of the protoplasmic processes in the deeper parts of the cell or by simple condensation of these fibrils to form the axis-cylinder, a perinuclear ring may be produced (fig. 8, pl. viii.). In this case the condensation may be irregular, and associated—by the passage along the nucleus of fibrils which, having sprung from the reticulum, are directed towards the axis-cylinder—with the formation of a perinuclear cap.

The most simple type of perinuclear condensation is that which is represented in fig. 16, pl. x. The reticulum is regular, and the condensation is derived from a thickening of the fibrils, and from a slight diminution of the size of the meshes.

Frequently in these cells some relation between the origin of the axis-cylinder and this perinuclear condensation can be recognised.

As I have already shown, the fibrils of the axis-cylinder may spring from that well-known zone of the cell which is called the cone, and which is left unstained in cells treated by Nissl's method, instead of starting from the border of the cell, without any structural character worthy of note.

The axis-cylinder fibrils situated in this zone may be connected with a condensation of the fibrillary reticulum, a condensation which, spreading further into the cell, may reach the perinuclear ring and be continuous with it. This peculiarity may be observed in fig. 3, pl. vi. Here also it may be seen that the axis-cylinder receives some of the long fibrils which run at the periphery of the cell. Even in cells in which the long fibrils are absent an analogous condition is met with; looking at fig. 7, pl. viii., a fine reticulum is seen to start from the perinuclear condensation. This is connected with the rest of the endocellular reticulum, and takes part in the formation of the axis-cylinder.

The zones of the reticulum which exhibit a thickening, and which are in communication with the perinuclear condensation on the one hand, and with the axis-cylinder on the other, have

different forms. They may spring from the cone of the axis-cylinder and taper off as they pass towards the perinuclear ring, and then terminate by forming a second cone opposite the cone of the axis-cylinder. This second cone is less dense than the other, but is more dense than the remainder of the endocellular reticulum. Or they may extend towards the centre of the cell, being continuous with a large portion of the perinuclear ring. These forms are met with most frequently in the cord and in the bulb.

The cells described occupy an intermediate position between the forms in which the axis-cylinder springs from the periphery, and those in which the axis-cylinder extends further into the cell and reaches with its long fibrils the centre of the cell, the perinuclear ring.

In the cells of the cerebral cortex, as I have already stated, it can be clearly demonstrated that the endocellular fibrillary reticulum takes part in the formation of the axis-cylinder (fig. 5, pl. vii.); but here I have not discovered the presence of a double cone, or of any special increase of density between the origin of the axis-cylinder and the perinuclear ring. Such formations are absent also in the cells of Purkinje. Why some cells should present this characteristic spreading of the perinuclear condensation towards the origin of the axis-cylinder and others not (fig. 2, and especially fig. 1, pl. v.) is not easy to determine. (Note that in fig. 1, pl. v., the axis-cylinder C, has been reproduced only in part; in the preparation it could be followed as far as the thinning down and the subsequent thickening, which is characteristic of the axis-cylinder; this structural change is seen in the prolongation marked *c* in fig. 4, pl. vi.; in figs. 5 and 6 in pl. vii.; in fig. 9, pl. ix.; and in the process shown in fig. 8, pl. viii.)

The forms already described are seen in cells in which the axis-cylinder fibrils are derived principally or even exclusively from the endocellular fibrillary reticulum, or from both this and from the long fibrils.

But, although rare, cells are sometimes seen in which the axis-cylinder fibrils take their origin chiefly from the system of long fibrils, as is shown in fig. 4, pl. vi. We cannot exclude that in the cell represented, the axis-cylinder receives some fibrils from the reticulum, but it is evident that in this case the bundle

of fibrils which lies along the right border of the cell supplies most of the fibrils to the axis-cylinder.

There exist types of cells also in which the axis-cylinder, instead of receiving fibrils from the periphery of the cell, passes further inwards, and obtains its fibrils from around the nucleus. But even in these cases the axis-cylinder may receive fibrils from the protoplasmic processes which have passed towards the deeper parts of the cell, as well as from the reticulum itself.

In fig. 9, pl. ix., there is a good example of such a formation : from the protoplasmic processes, *a*, *b*, *d*, come fibrils, which converge towards the nucleus, and after disappearing in the reticulum, they are collected together again into the axis-cylinder *c*. It is impossible to determine exactly, but it is probable that the perinuclear ring also takes part in the formation of this axis-cylinder.

In other cells, the axis-cylinder, although it takes origin from the deeper parts of the cell, collects also some fibrils from the peripheral long fibrils, as it passes out from the cell. An example of this is represented in fig. 6, pl. vii : focussing carefully with the microscope, the deep origin of the axis-cylinder is evident, and in the figure (although from faulty reproduction it is not so clear as one might wish) it is possible to see long fibrils passing into the axis-cylinder from the protoplasmic process on the left side.

The cell represented in fig. 8, pl. viii., demonstrates clearly how an axis-cylinder may obtain its fibrils from the endocellular fibrillary reticulum : this cell belongs to the type in which the axis-cylinder takes origin from the deeper portions of the cell. A considerable bundle of fibrils is present near the nucleus ; on the left, a zone may be seen, in which the beginning of the separation of the fibrils from the reticulum is suggested by the elongation of its meshes ; one can also see a smaller bundle passing around the nucleus from above, and going to form part of the fibrils of the axis-cylinder. Moreover, along its whole course through the reticulum, from near the nucleus to the periphery of the cell, the meshes of the reticulum are becoming elongated, and sending fibrils to the axis-cylinder, so that it is evident that this draws its roots, or collects its paths of conduction, not only from the perinuclear portion of the reticulum, but in great part from the remainder of the endocellular fibrillary reticulum.

We may infer, then, from these investigations, that the axis-cylinder may receive fibrils from the endocellular reticulum, or from this reticulum and, at the same time, and to a limited extent, from the long fibrils (peripheral, median or central); or, and this is rare, chiefly from the long fibrils.

Moreover, the starting-point of the axis-cylinder may be situated in different regions of the cell. It may spring from the periphery of the reticulum, or it may be in connection with a thickening of the reticulum, which may or may not reach the perinuclear condensation; or the axis-cylinder may arise from the deeper portions of the cell, and receive fibrils even from the level of the nucleus, entering into relation with the fibrillary reticulum and with the long fibrils, which may be found deep in the cell; or it may take origin chiefly, or it may be, exclusively, from the reticulum situated in the centre of the cell; or again, it may collect fibrils both from the deeper and the more peripheral portions of the cell; or it may show other modes of origin, which have their functional significance.

Now this different origin of the axis-cylinder fibrils, as regards their relation to the fibrils of the protoplasmic processes, and of the endocellular reticulum, cannot be a mere matter of accident.

It is quite evident, therefore, that the fibrils of the axis-cylinder take their origin from the endocellular reticulum, and that the seat of origin varies in the different cells, but is always demonstrable; the importance of this fact is obvious. It is on the demonstration of this fact that I have based the suggestion, which I have brought forward in some earlier publications, that the fibrillary reticulum, which can be exhibited by my methods, is an apparatus of a nervous nature.

As I have already said, my researches prove the existence of two types of cells: the first is characterised by cells provided only with an endocellular fibrillary reticulum, which is in relation with the fibrils of the protoplasmic processes; the second is more complex, and includes the great majority of the nerve cells. These possess two fibrillary systems, as I stated in 1900, and again at the International Congress of Physiology at Turin in 1901: (a) fibrils which form the endocellular reticulum; (b) fibrils which pass through the cell, but preserve their individuality. If we admit that the fibrils possess the function of con-



ducting nerve currents—*i.e.* “das leitende Element” of Apathy—because, according to my researches, there exists an evident relation of continuity between the endocellular reticulum and the fibrils of the prolongations, then it is fair to infer that the hypothesis—which I may add is only a hypothesis—which I expressed at the Congress at Turin, that the endocellular fibrillary reticulum is an apparatus of reception and of synthesis of the stimuli transmitted to it by the cellulipetal paths, is in all probability correct.

Employing this hypothesis, we may suppose that in the cells belonging to the first type mentioned above, only one system of conduction exists, that by which all the stimuli find their way to the reticulum, and that the axis-cylinder fibrils transmit only one set of currents, those which are derived from the endocellular reticulum.

In the second type of cells, however, we may infer two systems of conduction: one which has its centre in the endocellular fibrillary reticulum; the other, which corresponds to the fibrils of Bethe, which pass through the cell. When, therefore, in these cells the axis-cylinder is in relation with both of these systems of conduction (to a limited extent as regards the long fibrils, except in rare cases), it is possible to imagine that the axis-cylinder fibrils collect a double series of nerve stimuli, the most important of which are derived from the endocellular fibrillary reticulum, while the others, limited in number, are derived from the long fibrils.

To speak of cellulifugal and cellulipetal currents implies a hypothesis. The theory that the nerve stimuli travel along the fibrils of the axis-cylinder in a cellulifugal direction appears to be justified by facts which it is unnecessary to repeat. But, granted the cellulifugal function of the axis-cylinder fibrils, if the endocellular reticulum is admitted to be an apparatus for the reception of stimuli, it will be at once inferred that the fibrils of the protoplasmic processes possess a cellulipetal function.

But it is impossible to exclude that there are, in the protoplasmic processes, not only fibrils which conduct in a cellulipetal direction and which are connected with the endocellular reticulum, but also fibrils, which are equally connected with the endocellular reticulum, but which, at the same time, possess a cellulifugal function.

From the morphological point of view, it is certain that the fibrils of the protoplasmic processes enter into relation with the endocellular reticulum in various ways. Some go to form part of the reticulum adjacent to the point where the protoplasmic processes enter the body of the cell; others go to the central or perinuclear portion of the reticulum; others, again, to a zone intermediate between the two. Sometimes the reticulum extends for a greater or less distance into the protoplasmic processes themselves.

We have noticed frequently a condensation of the reticulum at the centre of the cell—perinuclear ring—(I may add that sometimes, but less frequently, a condensation is seen in the most peripheral portions of the cell). Now, the perinuclear condensation is very often in immediate relation with the origin of the axis-cylinder, either by a special condensation of the reticulum, which starts from the perinuclear condensation, and extends to the point at which the axis-cylinder fibrils emerge, or by the axis-cylinder fibrils springing directly from the deeper parts of the cell. These systems, which are distributed in the centre of the cell in the adult animal, I have seen also in the cells of the spinal cord in new-born animals. In fact, I have preparations made from tissues which have been taken from cats, which have been killed a few hours after birth, which illustrate these systems with great clearness, because they are more simple than in the adult, and because in some cells they represent the only system which is visible at this period.

In contrast to this condition, we find a morphological arrangement in which the axis-cylinder originates entirely from the periphery of the reticulum. If it be granted that, both when it has a central origin and when it has a peripheral origin, the axis-cylinder is always connected with the reticulum—which is a system in which anastomosis takes place—the position of the axis-cylinder would seem to be a matter of indifference.

But the morphology so often characteristic of the centre of the cell, the frequent presence of the condensation in close relation with the origin of the axis-cylinder, the not rare condition in which a large number of fibrils are found at the centre of the cell, are data which lead to the opinion that, at least in many instances, the central portion of the cell represents a zone of special importance, and that, therefore, the function of

the reticulum is not equally distributed throughout its whole extent.

At the same time, certain arrangements of the fibrils would seem to suggest a disposition adapted to render the transmission of the stimuli more rapid, as in the cases in which the axis-cylinder originates in the centre of the cell, and at the same time fibrils reach the centre of the cell from all the protoplasmic processes, and pass into the axis-cylinder, either directly or after joining the endocellular reticulum for a very short tract.

But, putting these hypotheses on one side, we cannot fail to recognise that the distribution and the various relations of the endocellular fibrillary reticulum must represent significant conditions with regard to the functions of the cell. However, that which interests us most at present is the morphological element, the precise investigation of the marvellous and complex structure of the nerve cell in the vertebrates. And it is more especially to the facts which are revealed in the endocellular fibrillary reticulum which I have described, and in the structures which have been the object of my researches, as well as the methods by which they may be exhibited, that I wish to direct the attention of students.

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Fig. 1

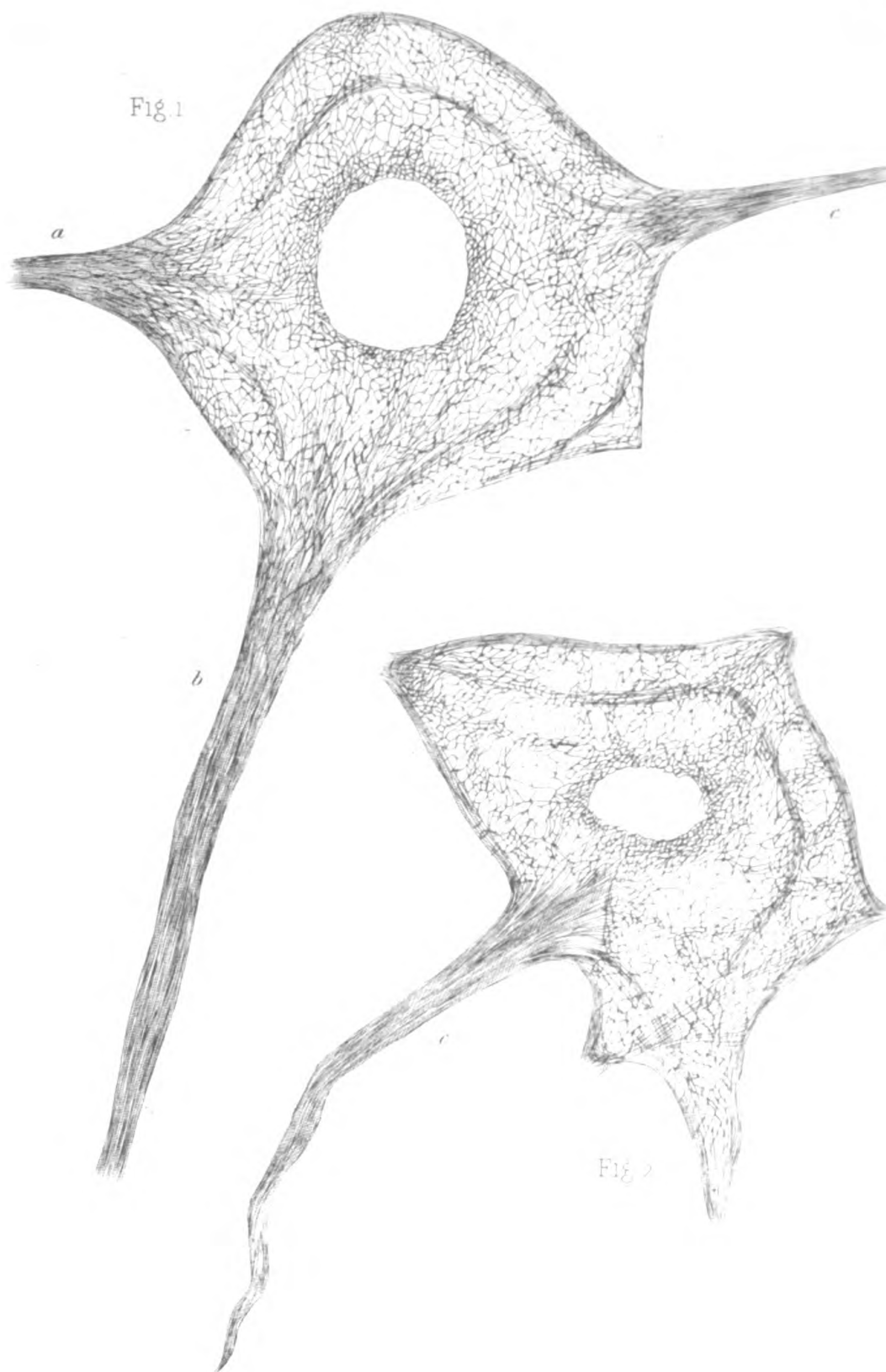


Fig. 2

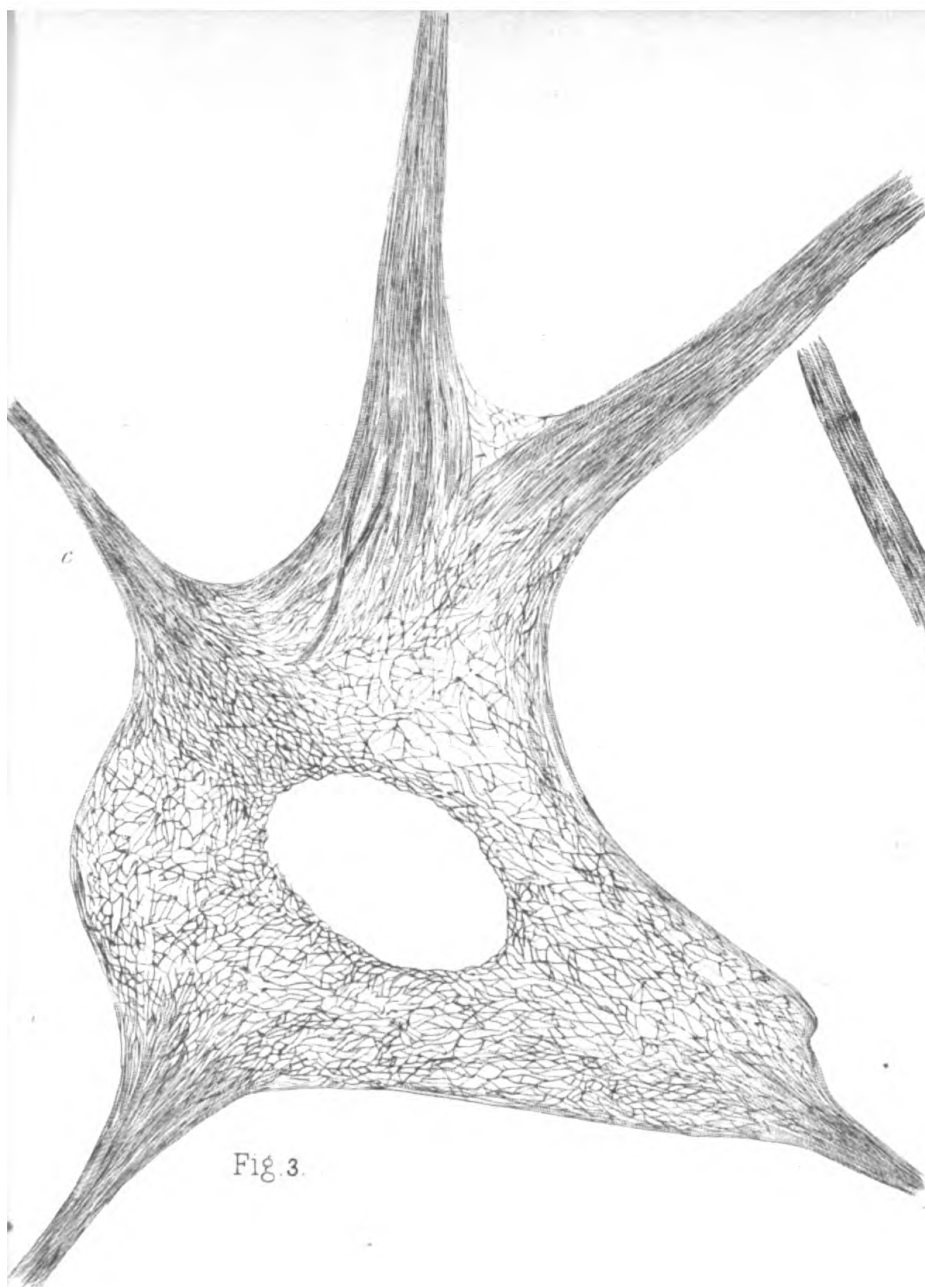


Fig. 3.

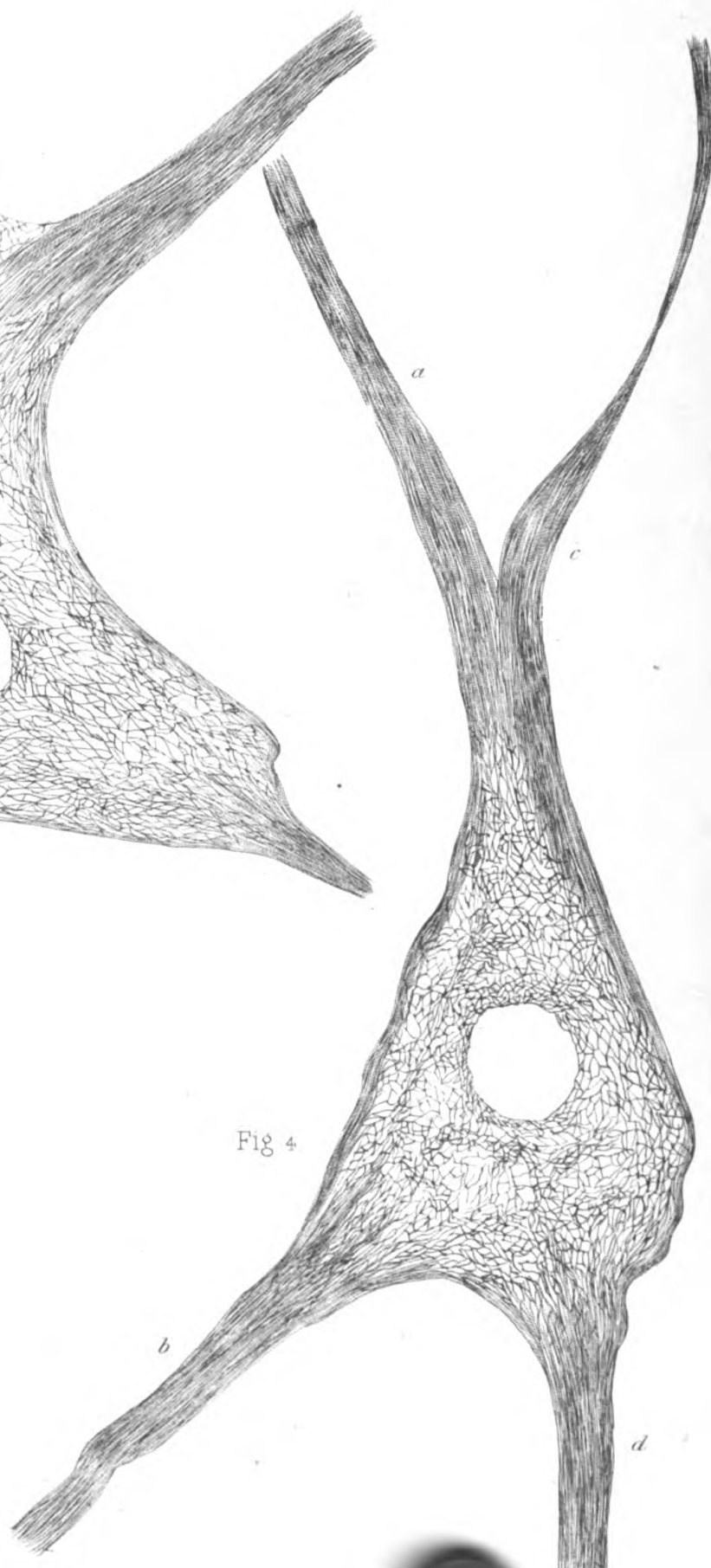


Fig. 4.







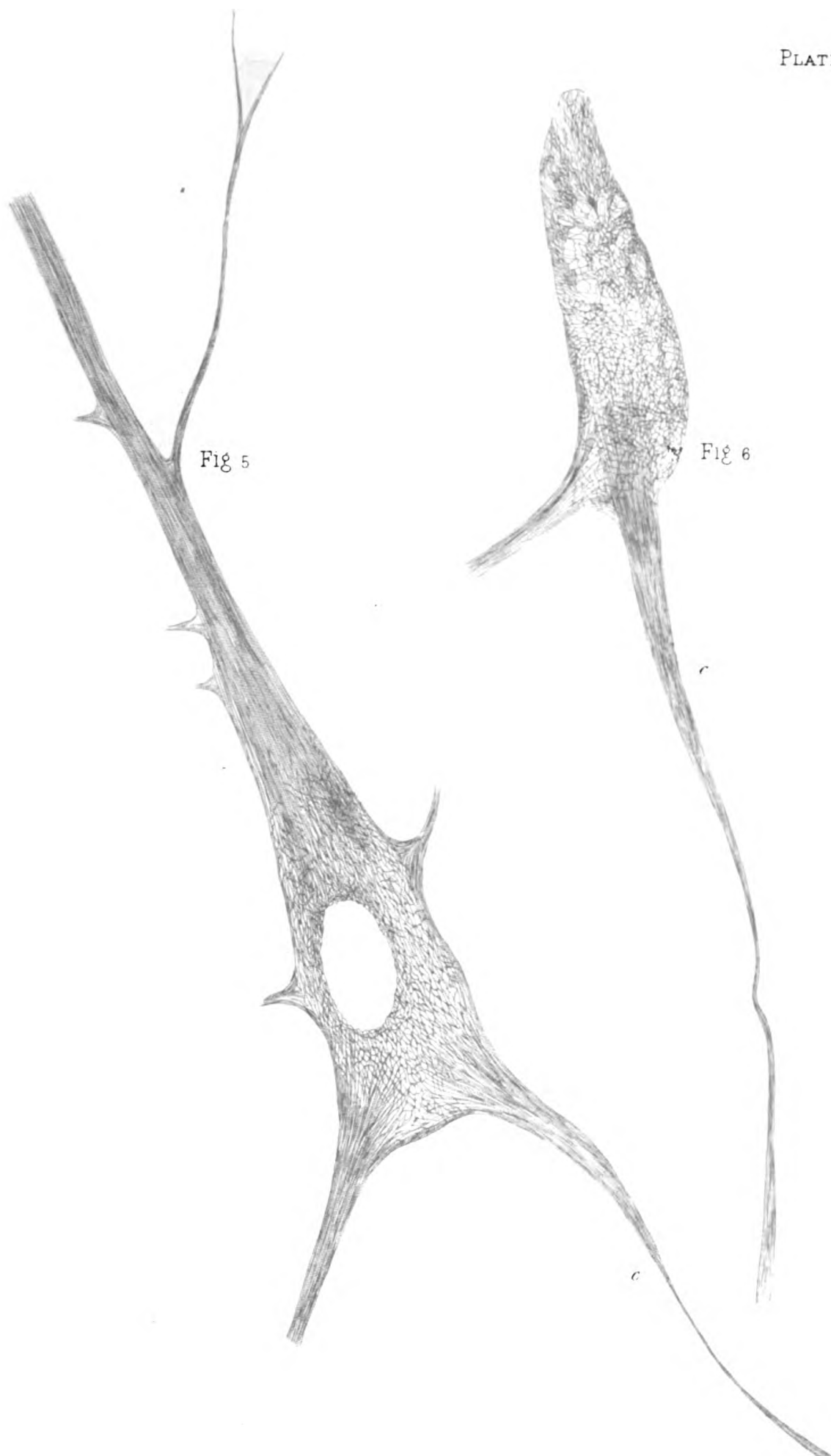


Fig. 7

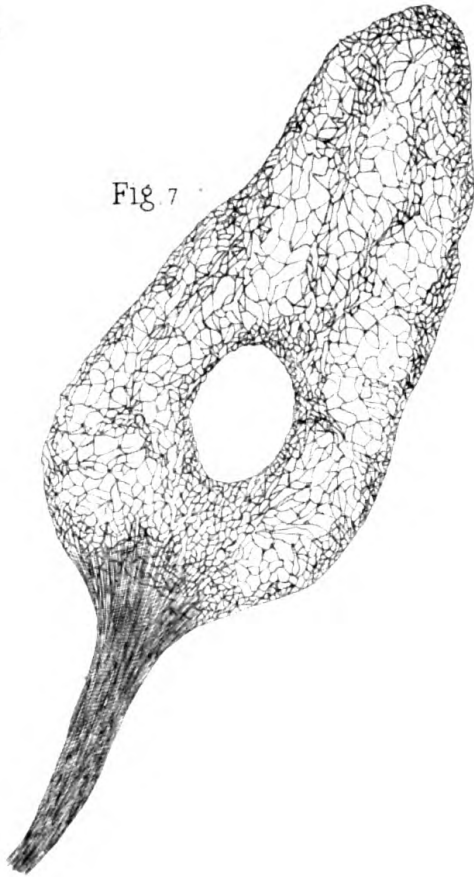
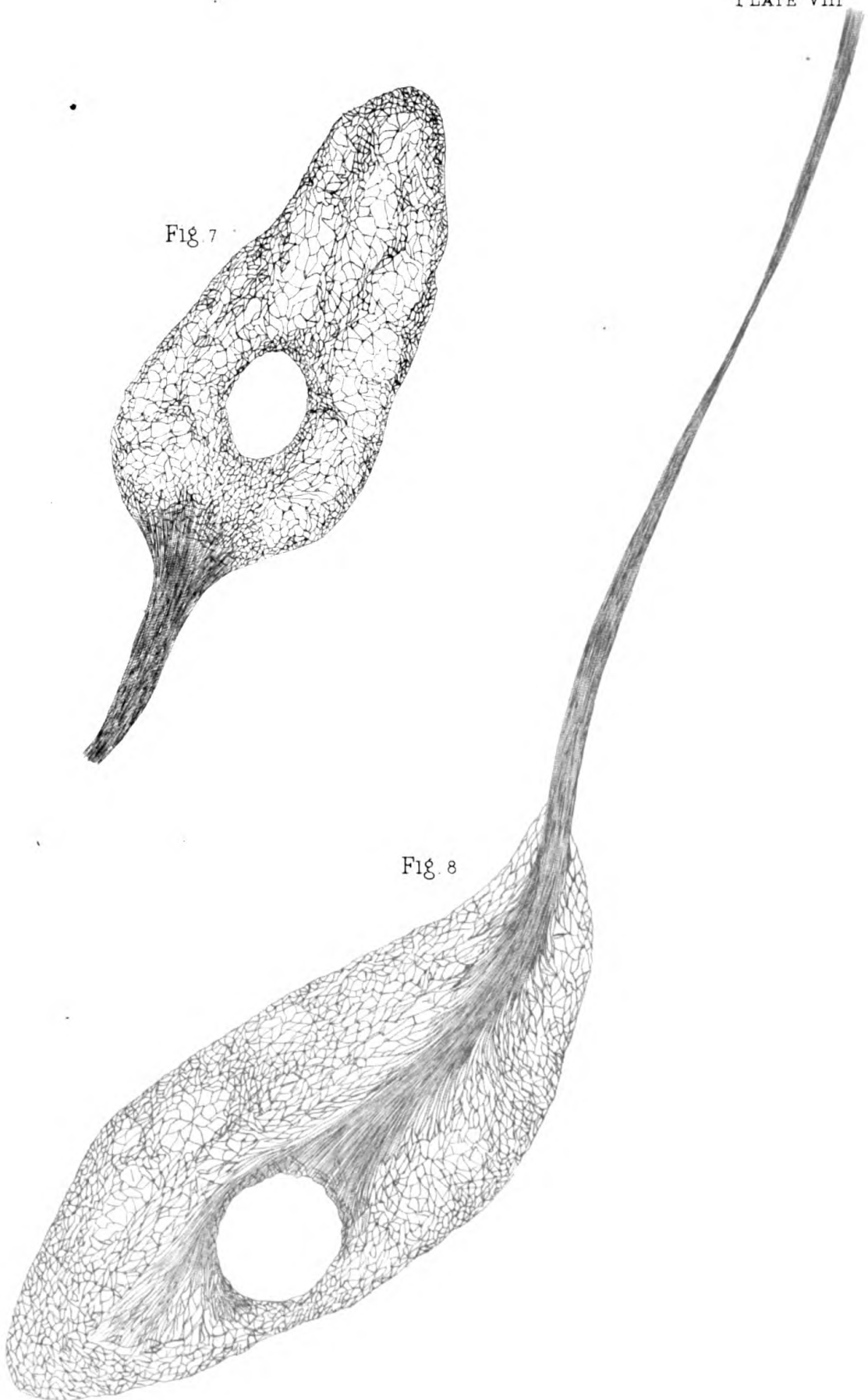


Fig. 8



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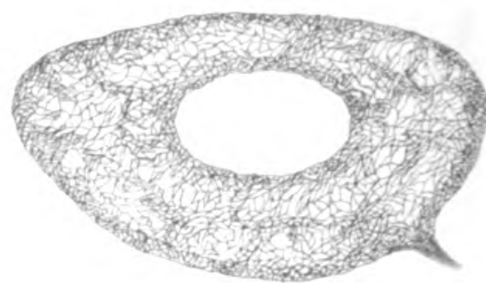
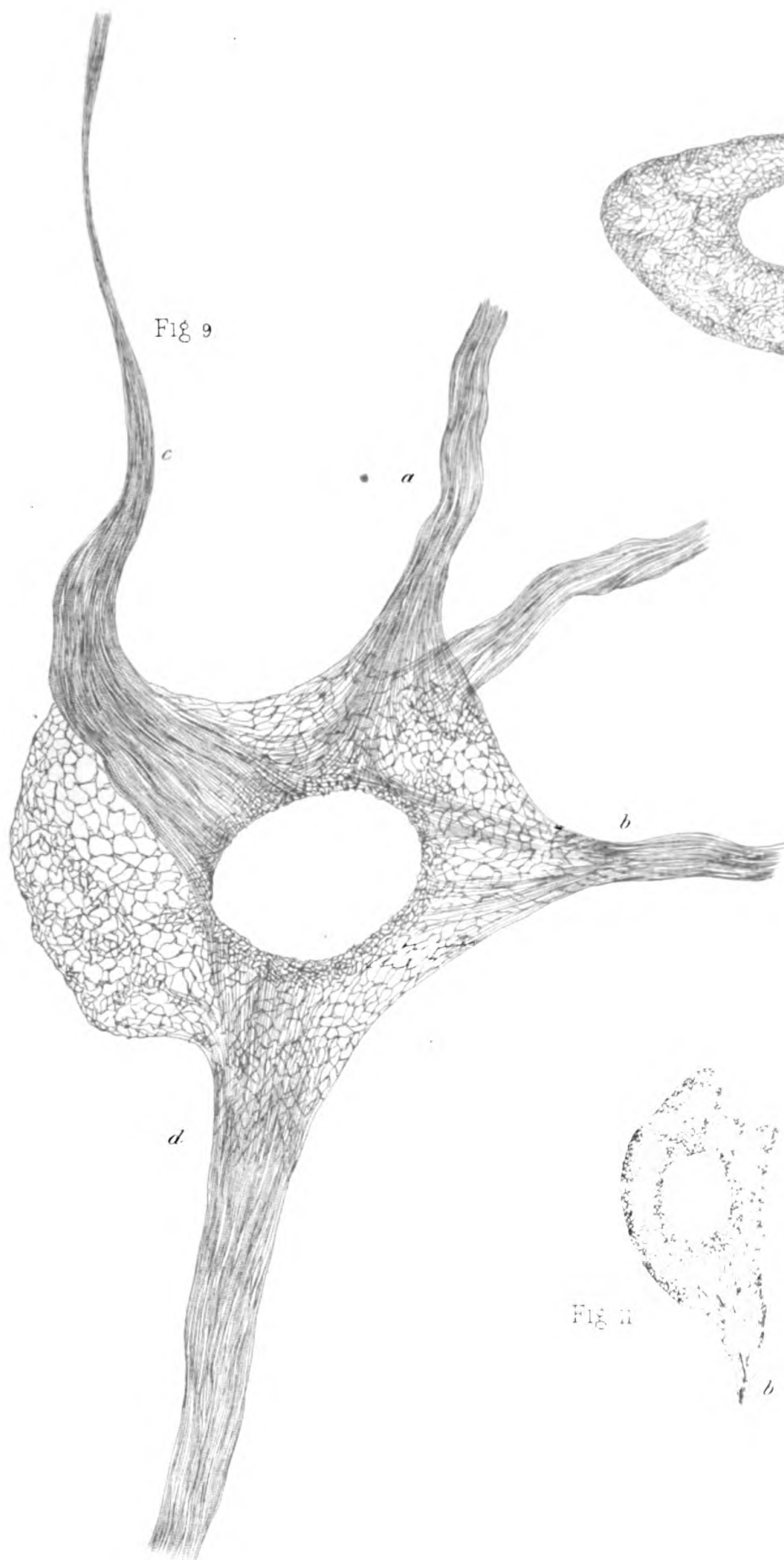




Fig 12

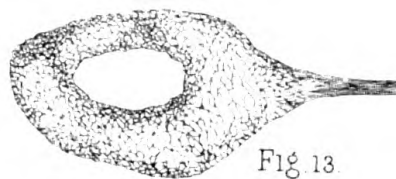


Fig 13

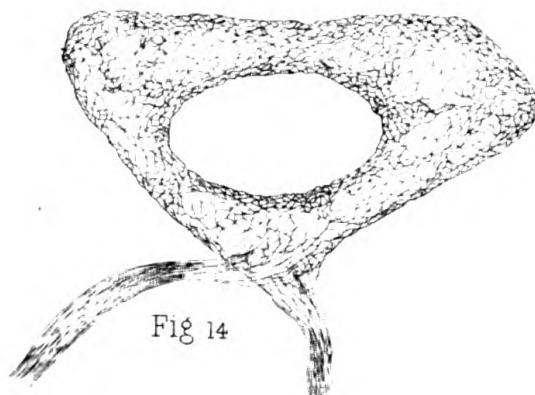


Fig 14



Fig 15

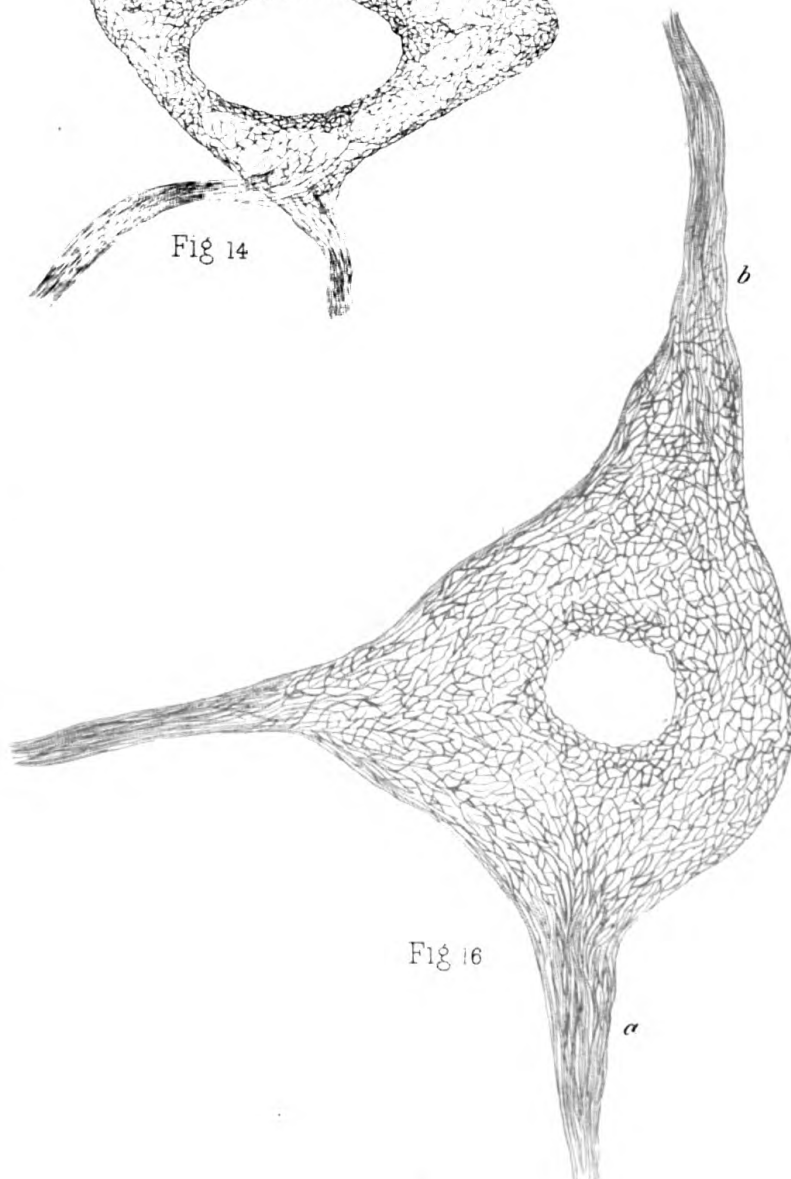


Fig 16



1902. *Gazzetta medica italiana*, anno liii., No. 45, p. 461. Vidi anche un sunto nel resoconto del Congresso pubblicato nella *Riv. sper. di Freniatria*, Vol. xxviii., fasc. 4, 1902; e sotto il titolo "Le fibrillenelle cellula nervosa," nella *Bibliographie anatomique*, fasc. 5., t. xii., 1903.

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17. A. Donaggio. Resoconto del 1° Congresso dei patologi italiani, nella *Gazzetta medica italiana*, No. 45, 1902; *Bibliographie anatomique*, fasc. 5, 1902; *Rivista sper. di Freniatria*, Vol. xxvii., fasc. 4, 1902.

#### † DESCRIPTION OF THE PLATES.

The sections 3, 4, 5  $\mu$  thick, have been examined with a  $\frac{1}{8}$  Leitz immersion lens—a, b, d = protoplasmic processes; c = axis-cylinder.

Figs. 1, 2, 3, 4, Pls. v. and vi. Cells from the anterior cornu of the spinal cord of an adult rabbit. In Fig. 3 the condensation of the peripheral ring extends to the origin of the axis-cylinder. Oc. 4, method 3 (pure pyridine).

Fig. 5, Pl. vii. Pyramidal cell from the cerebral cortex of an adult cat. Endocellular fibrillary reticulum and origin of axis-cylinder. Oc. 4, method 4 (pyridine and nitrate of pyridine).

Fig. 6, Pl. vii. Cell from the posterior cornu of the spinal cord of an adult rabbit. Oc. 4, method 3.

Fig. 7, Pl. viii. Medium-sized cell from the spinal cord of an adult rabbit. Condensation of the endocellular fibrillary reticulum between the cone of the axis-cylinder and the perinuclear ring. Oc. 12, comp. method 3.

Fig. 8, Pl. viii. Medium-sized cell from the spinal cord of an adult cat. Origin of axis-cylinder prolongation from the perinuclear portion of the



fibrillary reticulum, and from a long tract of the same reticulum. Oc. 12, comp. method 4.

Fig. 9, Pl. ix. Medium-sized cell from the posterior horn of the spinal cord of an adult rabbit. Origin of the axis-cylinder from the perinuclear position of the fibrillary ring and its relation with the fibrils of the protoplasmic prolongations, converging towards the centre of the cell. Oc. 12, comp. method 3.

Fig. 10, Pl. ix. Small cell from the posterior horn of the spinal cord of an adult rabbit. Close fibrillary reticulum with condensation. Oc. 8, method 3.

Fig. 11, Pl. ix. Id. A bundle from the protoplasmic process (b) is taking part in the fibrillary reticulum. Oc. 4, method 3.

Fig. 12, Pl. x. A very small cell from the posterior horn of the spinal cord of an adult rabbit. Endocellular fibrillary reticulum with slight perinuclear ring. Oc. 4, method 3.

Fig. 13, Pl. x. Small cell from the posterior horn of the spinal cord of an adult guinea-pig. Perinuclear ring. Oc. 4, method 3.

Fig. 14, Pl. x. Cell from the posterior horn of the spinal cord of an adult rabbit. Perinuclear ring. Oc. 4, method 4.

Fig. 15, Pl. x. Small pyriform cell from the cerebral cortex of an adult dog. Uniform fibrillary reticulum; slight perinuclear ring. Oc. 4, method 4.

Fig. 16, Pl. x. Cell from the anterior cornu of the spinal cord of a dog aged one month. Slight perinuclear ring; in (b) the reticulum extends rather far into the protoplasmic process; the fibrillary reticulum has a uniform aspect. Oc. 4, method 3.

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### SOME ASPECTS OF ALCOHOLISM.

By A. HILL BUCHAN, M.A., M.B., M.R.C.P.E.

(Continued from p. 30.)

OF the above series of cases of D.T., 20 men and 6 women showed symptoms of peripheral neuritis. It is proposed at this point to take up all the neuritis cases in the records, as well as those occurring along with D.T.

Out of a total of 418 cases of alcoholism, 62 had definite symptoms of neuritis. While the condition of many patients did not permit a detailed examination, the above figures are probably not far from correct. It is remarkable how frequently half-conscious patients, who scarcely respond or have ceased to respond to other stimuli, manifest signs of pain when pressure is made along the course of nerves most affected.

*Sex.*—There is a striking contrast between the sexes in relation to this disease. While of the total 418 cases of

alcoholism, 350 were men and 68 women, of those which had neuritis, 38 were men and 24 women. In other words, while women constitute only 16·3 per cent. of the total admissions, they form 38·7 per cent. of the neuritis cases.

*Age.*—The average age of male patients was 40·0, of female 40·4.

TABLE XII. CASES ARRANGED ACCORDING TO AGE,  
IN FIVE-YEARLY PERIODS.

	21-25	26-29	30-35	36-40	41-45	46-50	51-55	56-60	61-65	66-70	71-75
Male . . .	0	5	8	7	5	6	1	1	...	1	2
Female . . .	2	4	2	5	3	3	2	...	1	...	...
	2	9	10	12	8	9	3	1	1	1	2

Thus the larger number of cases occur before the age of 40-50, and only a very small proportion before 25. Though no case of neuritis was met with in a man under the age of 28, there were three cases among the women below that age, viz., one at 22, one at 23, and one at 27.

While the average ascertained duration of alcoholic habits was 14·3 years in the case of the men, in that of the women it was 12·2 years.

TABLE XIII. SHOWING DURATION OF ALCOHOLIC HABITS.

	1-1	1-2	2-3	3-4	4-5	6-10	11-15	16-20	21-25	26-30	31—Years
Men . . .	1	2	0	0	1	3	2	5	1	1	1
Women . . .	0	1	2	1	0	0	2	0	0	1	1
	1	3	2	1	1	3	4	5	1	2	2

TABLE XIV. SHOWING DURATION OF BOUT PRECEDING PATIENT'S  
ADMISSION TO WARD.

	NUMBER OF WEEKS.						
	1	1½	2	3	4	6	8
Men . . .	1	1	2	2	3	2	2
Women . . .	2	...	...	1	...	1	1

*Heredity*—in the small number of cases where it could be ascertained—is shown in the following table :—

TABLE XV.

Case	Family History of Alcoholism.	Family History of Insanity.	Family History of other Affections of Nervous System.
1	Mother and brother.		Sister.
2	Father, mother, brother and sister.	Brother.	
3	Uncle (maternal) and brother.		
4	Father and two brothers.		
5	Father and mother.		
6	Father and mother.		

*Occupations of patients.*—Baker, 1 ; beer bottler, 1 ; bottle-blower, 1 ; bookkeeper, 1 ; butler, 1 ; cabmen, 2 ; clerks, 4 ; clergymen, 1 ; compositor, 1 ; constable, 1 ; cricketer, 1 ; dairyman, 1 ; hawker, 1 ; house-painter, 1 ; housewives, 4 ; lodging-house keeper, 1 ; publican, 1 ; rubbermaker, 1 ; servants, 2 ; sheep farmer, 1 ; stableman, 1 ; washerwoman, 1 ; wheelwright, 1.

*Form in which alcohol was taken.*—In 19 cases where a note has been made regarding this we find :—

Whisky in 18 of these cases.

„ alone in 6 of these cases.

„ with beer in 7 of these cases.

„ with porter in 1 of these cases.

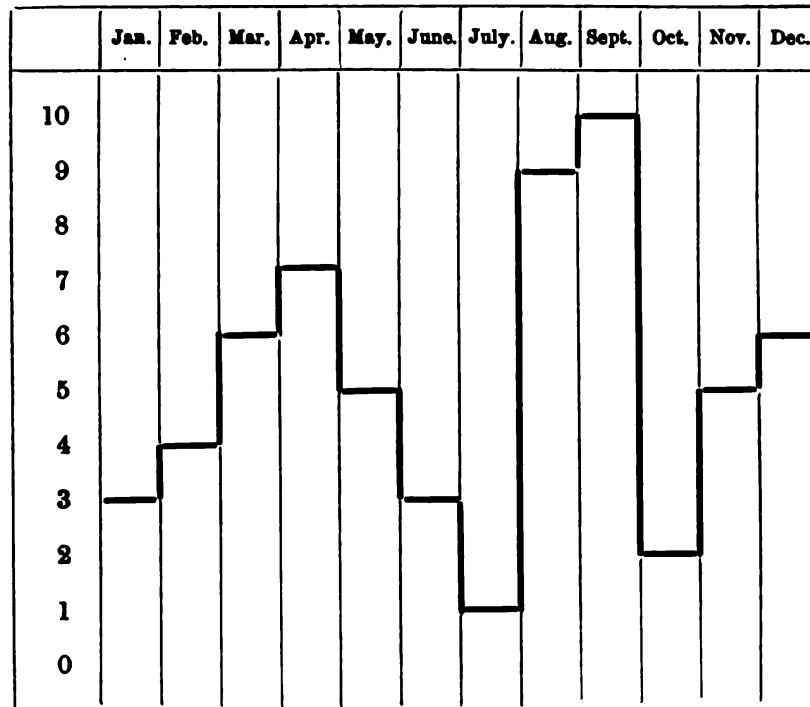
„ with porter and sherry in 1 of these cases.

In no case is it recorded that beer was the only drink.

*Amount of alcohol consumed daily.*—As far as the notes give this, we find the following for individual patients: 3 glasses whisky; 1 pint whisky; 3 to 5 glasses whisky; 1 gill whisky and 5 glasses beer; 1 bottle whisky;  $\frac{1}{2}$  pint whisky; 1 bottle whisky and several pints beer.

*Season.*—

TABLE XVI. SHOWING SEASONAL OCCURRENCES.



*Previous illness.*—Ten cases had previously suffered from D.T. or had “been in the ward”; two had had influenza, in one the attack having occurred 6 weeks before patient’s admission; 3 had had rheumatism; 2 syphilis; 3 fits; 1 had attempted suicide; 1 had a history of “ague.”

*Circulatory System.*—The average highest pulse rate was 101.

TABLE XVII. HIGHEST PULSE RATES.

	70-80.	81-90.	91-100.	101-110.	111-120.	121-130.	131-140.
No. of cases .	6	8	14	11	11	3	0

TABLE XVIII. TEMPERATURE.—HIGHEST READINGS.

	98-100	100-1-101	101-1-102	102-1-103	103-1-104	104-1-105	105-1—
Jan.	4	...	...	...	...	...	...
Feb.	3	1	...	...	...	...	1
March	3	1	1	...	...	...	...
April	2	...	1	1	1	1	1
May	1	1	...	...	...	1	...
June	1	...	...	1	...	...	...
July	1	...	...	...	...	...	...
Aug.	6	...	...	...	...	1	...
Sept.	8	...	...	...	...	...	...
Oct.	1	...	...	...	...	...	...
Nov.	5	...	...	...	...	...	...
Dec.	3	1	...	...	2	...	...

*Nervous System.*—The frequency of various subjective sensations complained of was as follows:—

Numbness	.	.	.	.	22
Pain	.	.	.	.	19
Tingling	.	.	.	.	9
Cramps	.	.	.	.	10
Feeling of excessive cold	.	.	.	.	7
"Tired" or "sore" feeling	.	.	.	.	2
Feeling of excessive heat	.	.	.	.	1
Giddiness with eyes shut	.	.	.	.	1
Double vision	.	.	.	.	1

*Sensibility.*—

(α) To tactile impressions, entered as normal in 19 cases.

" " " delayed in 3 "

" " " impaired in 9 "

(β) To painful stimuli.—

(1) *Superficial* or cutaneous.—

Hyperalgesia present in 10 cases.

Complete or partial analgesia present in 3 cases.

Delay in perception of pain present in 3 cases.

Entered "no delay" in 2 cases.

Stimuli perceived first as tactile, then later as painful impressions in 1 case.

"Normal" present in 6 cases.

(2) To *deep* pressure on muscle and nerves.—

Hyperæsthesia present in 62 cases.

(γ) Temperature sense, entered as normal in 15 cases.

Sensibility to heat impaired in 5 cases.

„ „ delayed in 5 cases.

„ „ increased in 1 case.

„ to cold impaired in 3 cases.

„ „ delayed in 1 case.

„ „ increased—none noted.

Cold was more readily felt than heat in 7 cases.

Heat „ „ cold in 0 cases.

Loss or impairment of ability to distinguish heat from cold in 4 cases.

(δ) Muscular sense and co-ordination.—

The tremor movements of the patients render investigation as to the occurrence of ataxia difficult. In some cases of the present series it appeared to be present.

Sense of position is noted as normal in 7 cases.

„ „ „ defective in 2 cases.

Unsteadiness with eyes shut present in 8 cases.

In one patient special reference is made to the fact that he could stand steady with eyes open, while he tended to fall to the left side if they were shut.

Of one patient it is stated that he required as much support when standing with eyes open as shut; that he could place his foot in a given position, but that there was inaccuracy in movement when he attempted to point with feet or hands.

In one patient flexion and extension at the knee though powerful was interrupted, as if some inco-ordination was present. There was also some inco-ordination as he raised or put down his foot, and after walking a few yards the legs seemed to get beyond his control.

*Special Senses.*—Investigation too incomplete to render any statement of value.

#### RELATION OF SENSORY TO MOTOR DISTURBANCES.

	With Sensory Symptoms only.	With Motor Symptoms only.	With both Sensory and Motor Symptoms.
Male,	9 cases.	1 case.	22 cases.
Female,	14 cases.		11 cases.

It is often more difficult to exclude the absence of motor than of sensory defects.

## REFLEXES.

(α) *Organic*—

In 23 cases they are entered as normal.

In 2 micturition defective.

In 4 there was loss of control of defæcation.

Pupils were "equal" in 5; "unequal" in 5; of "medium size" in 5; "dilated" in 3; "small" or "contracted" in 7. Reaction to light notes as present in 12, as sluggish in 1.

Reaction to accommodation is present in 11, sluggish in 1.

"Reaction normal" in 12 cases.

(β) *Superficial*—

	Plantar.	Crema- steric.	Abdo- minal.	Epi- gastric.
"Present" or "normal" in	6	4	3	3
Increased . . .	19	...	2	1
Diminished . . .	4	1	2	...
Lost . . .	2	2	3	3

(γ) *Deep*—

	Ankle Clonus.	Knee Jerks.	Biceps Jerks.	Triceps Jerks.	Adductor Jerk.
"Present" or "normal"	...	15	...	...	...
Increased . . .	4	15	...	...	Slightly.
Diminished . . .	...	6	1	...	...
Absent . . .	19	8	1	2	...

One notices the frequency of occurrence of increase in the plantar reflex. But the most interesting point is the large number of instances in which the knee jerks were either present or increased.

An explanation of this is most likely to be found in the fact that many of the cases came into the wards during the early stage of the disease, which was in the sensory form without muscular implication.

*Motor power.*—

Upper limbs	{	Drop wrist present in 6.
		Extensors of arm impaired in 1.
		Paresis in 4.
Lower limbs	{	Drop ankle present in 7.
		" absent in 6.
		Loss of elasticity of gait without definite "paralysis," 4.
		Inability to stand on toes, 1.
		Paresis or paralysis, 15.
		"No paralysis," 6.

One patient was unable to sit up or to turn in bed. Two had paresis of external eye muscles.

There is thus a preponderance of affection of the lower over the upper extremities and of paralysis of the extensors over the flexors.

The following table shows the number of instances in which departure from normal was noted in one or other region :—

TABLE XIX.

	Subjective feelings.	Tactile sensibility.	Pain sensibility.	Temperature sense.	Conductive.	Vol. power.	Atrophy.	Tremor.
Total number of cases in which—								
Lower extremities in whole or part affected . . . . .	28	20	50	2	12	24	...	1
Thighs affected or "specially affected" . . . . .	2	4	17	...	...	2	...	...
Legs do. do. . . . .	25	19	42	2	...	8	...	1
Legs affected or "specially affected," of these "specially calves" . . . . .	5	10	31	...	...	1	...	...
Feet affected or "specially affected" . . . . .	7	1	5	1	...	...	...	...
Upper extremities in whole or part affected . . . . .	8	11	21	1	...	8	...	1
Upper arm affected or "specially affected" . . . . .	1	1	1	...	...	1	...	...
Forearm do. do. . . . .	...	...	9	1	...	6	...	...
Hand and fingers do. . . . .	8	1	2	1	...	4	...	9
Trunk affected . . . . .	2	2	5	2	...	1	...	...
Lumbar region affected or "specially affected" . . . . .	2	...	1	...	...	...	...	...
Back do. do. . . . .	1	...	...	1	...	...	...	...
Lower part of abdomen affected or "specially affected" . . . . .	...	1	...	...	...	...	...	...
Pectorals do. do. . . . .	...	...	2	...	...	...	...	...
Head do. do. . . . .	2	...	...	...	...	...	...	...
Face do. do. . . . .	...	1	...	...	...	...	...	...
Infra-clavicular region do. do. . . . .	...	...	...	1	...	...	...	...
Lips do. do. . . . .	...	...	...	...	...	...	...	2
Tongue do. do. . . . .	...	...	...	...	...	...	...	10
Eye extrinsic muscles do. do. . . . .	...	...	...	nystagmus	2	2	...	2
"All over body" do. . . . .	1	2	2	...	...	...	...	3



The most frequent departure from the normal was disturbance in regard to pain in lower extremities, this being almost always in the form of muscular hyperæsthesia. The almost invariable seat of pain on pressure was in the calves. It was very frequently found that during the acute stage of an alcoholic attack the calves showed a degree of sensitiveness to pressure, but when this passed off in a few days the case was not regarded as being one of peripheral neuritis, but only so when the tenderness persisted.

Practically through the whole series one finds the same preponderance of affection of the lower over that of the upper extremities, as also of the posterior over the anterior aspects of the limbs.

It will be observed that in several cases the inner aspect of the lower extremities was more affected than the outer, as seen in

TABLE XX.

CASE	TACTILE SENSIBILITY	SENSIBILITY TO PAIN	
		Superficial	Deep
1	...	Analgesia inner side thigh	...
2	...	Tenderness above int. condyle	...
3	...	Analgesia inner side legs	...
4	...	Tenderness inner aspect legs and foot	Esp. adducts or thigh
5	...	Tenderness ext. poplit. nerves and ant. tibial	Ext. popl. nerves
6	Strip partial anæsthesia inner aspect legs	...	...
7	Anæsthesia inner aspect legs, anæsthesia below knee, except on sole foot and a little on dorsum rt. foot near outer border	...	...
8	...	Analgesia extending above lower half thigh on inner side	...

In no case is there noted the very acute form of superficial hyperalgesia sometimes present in neuritis.

*Mental Symptoms.*—The next table shows these arranged according to sex and result. The term recovery is applied to all cases in which such improvement occurred as to admit of the patients being sent home, such marked mental disturbances as, e.g. hallucinations, having ceased; but is not meant to imply

that a complete restoration to former mental state had been effected. The ward being only for acute cases, the records do not afford data as to the latter point, nor as to the full course of the neuritis.

TABLE XXI. MENTAL SYMPTOMS.

	MALE			FEMALE		
	Recovered	Sent to Asylum	Died	Recovered	Sent to Asylum	Died
Duration of mental symptoms (average being estimated from day of admission) . .	3 days	...	...	3.5	...	...
Average number of days after admission on which first sleep occurred . . . .	1.3	1	...	1.2	...	...
Persistence mental disturbances after sleep . . . .	4	...	2	2	1	...
Illusions . . . . .	1	...	1	1	...	...
Hallucinations . . . . .	12	2	3	3	1	...
Hallucinations of vision . .	12	...	...	3	1	...
Hallucinations of hearing . .	10	...	...	2	1	...
Delusions . . . . .	8	1	1	1	3	1
Hallucinations or delusions relating to animals . . . .	4	1	...	1	...	...
Delirium about occupation . .	4	...	...	1	...	...
Delusions of persecution . .	3	...	2	2	2	...
Ideas of grandeur . . . . .	0	...	...	...	...	...
Ideas of fear . . . . .	3	1	...	1	...	...
Systematised delusions . . .	3	...	...	...	2	...
Explanatory delirium . . . .	3	...	...	...	...	...
Suicidal tendencies . . . . .	2	...	...	1	1	...
Excitement specially marked	4	...	...	2	1	1
Memory impaired for distant past . . . . .	2	...	...	...	2	...
Memory impaired for near past	3	...	...	1	2	1
Orientation impaired for time	5	...	...	2	2	...
Orientation impaired for place	6	...	...	1	2	...
Coma or lethargy . . . . .	1	...	1	2	...	3

*General Result.*—31 cases are entered in the records as having recovered; 13 as improved; 7 were sent to asylum; 3 transferred to a general ward; 8 died.

Dr Septimus Reynolds at a meeting of the Medico-Chirurgical Society of London (*Med. Chir. Soc. Lon. Transactions*, vol. lxxxiv., p. 409) on Jan. 8, 1901, in his paper on the recent epidemic of arsenical neuritis in England, threw out the suggestion that all cases of "so-called alcoholic neuritis" might be really due not to alcohol, but to arsenic present in the drink. The series of cases at present under review does not appear to give support to Dr Reynolds' theory. Dr Reynolds says that the aspect of the

patients with arsenical neuritis was so typical that their cases could generally be diagnosed at sight. But the puffiness of the face, suffused and watery eyes, crimson-red colour of face, husky voice, etc., which were among the features characteristic of arsenical neuritis, were not once noted in the records in hand, and had they been present their existence could not during so long a period have remained unrecognised. The same holds true regarding erythromelalgia, keratosis, erythemata, acute urticaria, pigmentation, herpes zoster, loss of hair, affections of nails so common in the recent epidemic.

Atrophy was noted as a very marked feature in some of the arsenical cases—apparently quite out of proportion to the severity of the neuritis; the hands in some cases closely resembling what one is accustomed to see in progressive muscular atrophy. No such remarkable disproportion in this respect is noted in the Edinburgh records.

Ataxia, which we have seen was not a prominent symptom here, was very noticeable in the epidemic.

As to motor paralysis, Dr Reynolds says that the symptoms were similar to those found in "so-called alcoholic neuritis"; but, as pointed out by Dr Judson Bury (*Med. Chir. Soc. Lond. Transactions*, Vol. lxxxiv., p. 437)—and our records accord with the statement—in a case of alcoholic paralysis, the extensors of the wrist and flexors of the ankle are predominantly affected. Arsenic, on the other hand, appears to pick out and attack more severely the extensors of the fingers and toes.

Dr Reynolds refers to the affection of the small muscles of the hand. Twenty-five per cent. of Dr Reynolds' cases showed oedema—much beyond the percentage in our series. In Dr Reynolds' cases, cramps when present were often in the feet, and especially about the big toe rather than in the calves, as in our series.

The cases of arsenical neuritis were almost invariably beer-drinkers. A few had confined themselves to stout. Kelynack and Kirkby say that they were unable to meet with evidence of arsenical poisoning in any persons limiting themselves to the use of brandy or whisky. "It is a remarkable fact," they go on to say, "that even in confirmed drinkers in this district who have restricted themselves, according to their own account, to spirit, peripheral neuritis is so rare that we are not prepared to substantiate a single case met with in recent years."

The authors do not inform us what proportion of their alcoholic patients limit themselves to brandy or whisky.

Now, there can be no question that in Edinburgh whisky is the form in which alcohol is mostly taken.

One may draw attention to the six cases above in which whisky was the only drink.

Comparing the cases of alcoholism in Edinburgh with those reported during the recent arsenical poisoning epidemic, one notices that, along with having common features, there was a definite absence in the former of certain symptoms prominent in the latter; and without some further evidence one cannot look on the disease we have been accustomed to describe as alcoholic neuritis as really of arsenical origin.

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**A MICRO-CHEMICAL EXAMINATION OF THE PHOSPHORUS IN BLOOD-CLOTS : an Attempt to differentiate between Clots formed during Life, after Death, and those artificially produced by Alcohol used in preparing Tissues for Sections.**

By JOHN TURNER, M.B. Aberd.

IN the course of some investigations into the pathological anatomy of epilepsy, it became of the highest importance to determine whether clots formed in the vessels of the nervous system had been formed during life, or were the result either of the fixative used in preparing the tissues for sections, or of post-mortem changes.

This led to a series of experiments with blood-clots, which were tested micro-chemically for phosphorus by Professor Macallum's phenyl-hydrazin hydrochloride test, in order to see where the phosphorus was located under different circumstances, and whether by this means the problem could be solved.

The theory which holds the field at present concerning blood coagulation is, that under normal conditions the leucocytes, the blood-plates, and to a lesser degree the red corpuscles, contain nucleo-proteid. These elements under pathological conditions, as, for example, when blood is shed, or a vessel injured, get rid of

their nucleo-proteid contents, which combine with the lime salts of the plasma to form fibrin ferment, and this again combines with the fibrinogen of the plasma to form fibrin. In this process, the formation of fibrin, Professor Halliburton says, is "the essential act of coagulation."

Now, nucleo-proteid contains phosphorus, and therefore the fibrin formed in the way just described will contain phosphorus, and should stain green with Macallum's test, and so it does.

Lilienfeld and Monti were, I believe, the first to introduce a test for the detection of phosphorus in the tissues, for which purpose they employed ammonium molybdate and pyrogallie acid. The method, according to Macallum, is not altogether to be depended upon, and in 1897 he introduced another, in which tissues fixed in alcohol are subjected to the action of molybdate of ammonia in nitric acid, and subsequently treated with a solution of phenyl-hydrazin hydrochloride. By this means phosphorus-containing material is stained green. This, with some slight modifications, is the method which I have employed.

Macallum places the tissue, after hardening in alcohol, in a Soxhlet apparatus, which extracts the phosphorised fats. This part of his procedure was omitted in my observations, and the test was employed in this manner:—Pieces of nervous tissue were fixed in absolute alcohol for twenty-four hours, then passed through chloroform, infiltrated with paraffin, sections cut, fixed to the slide by Gulland's method, the paraffin extracted as usual. The slide washed in distilled water, and placed for eighteen hours in a mixture made by dissolving 1 part of pure molybdic acid in 4 parts strong ammonia, and adding 15 parts of nitric acid, S.G. 1.2. After this they are mixed in nitric acid or distilled water, and a freshly prepared 1-4 per cent. solution of phenyl-hydrazin hydrochloride is poured over them and left for a few minutes, washed, dehydrated, cleared in xylol, and mounted in balsam.

The fresh blood films were merely dried and then placed in the molybdic mixture, the further treatment being the same as in the case of the sections.

A more or less firm, white, ante-mortem clot is taken from a corpse, sections cut of it, and tested for phosphorus. In the course of a few seconds they begin to colour a blue-green, and

in four or five minutes this colour has intensified, and they are ready to be examined microscopically. Their structure consists chiefly of fibrin threads, varying in thickness, some as much as 2 m., others too thin to measure accurately. These threads lie in all directions, forming a fairly coarse meshwork, in the spaces of which lie leucocytes and blood-plates. All these structures are stained green, the fibrin lightest, the blood-plates darkest.

The blood-plates are a prominent feature, especially in those parts of the clot where the fibrin threads are coarsest. These threads would appear to be the more recently formed, and therefore the less organised parts of the clot. Scattered about are dark-green masses from which radiate fibrin threads. It is difficult to make certain whether the nucleus of these consists of a leucocyte or a cluster of blood-plates. In some cases the appearances are in favour of the former view, in others of the latter. Very few red corpuscles are present, and they are generally to be found at the edge of the clot. Some stain very pale green, others yellow. Speaking generally, the more recent the clot, the more apparent are the blood-plates and certain larger bodies which probably consist of fused blood-plates. In these recent clots a quantity of finely granular debris is seen staining pale green, and this in all probability represents a disintegration of the blood-plates.

The clot which forms in fluid blood taken in considerable quantities from a corpse a few hours after death, presents in the main, appearances similar to those just described; but in all probability, although this clot is formed after the death of the subject, it represents the vital activity of blood-cells which are still living.

Dark red, soft post-mortem clots taken from the heart at various periods after death were treated similarly.

The sections coloured a darker, more yellow green and more quickly than in the case of ante-mortem clots. When examined microscopically, however, this green colour was found to be entirely confined to the blood-elements, the darkest coloured of which were the blood-plates, and the masses into which they fuse, and the red cells (of which the section chiefly consists) were the palest. Some of the leucocytes showed fibrin threads radiating from them, otherwise there was no fibrin to be seen; but amongst the cells was a quantity of yellow homogeneous material, forming little islands, with occasionally a

green stained leucocyte imbedded in their midst. This yellow material evidently formed the clot.

When unclotted blood, as, for example, that which is contained in the vessels of the sections of the brain substance, is examined, the red corpuscles stain of a very pale green with a darker rim—in fact, quite similarly to the majority of those found in post-mortem clots; and the leucocytes and blood-plates similarly are darker green.

If a drop of blood from the living subject is allowed to fall on a slide, and spread out to form a thick film—dried at room temperature and tested for phosphorus—it slowly assumes a dull olive green colour, and when looked at under the microscope the red corpuscles are seen to be of a reddish yellow without a trace of green; they lie in a blue-green homogeneous bed in which also the leucocytes and blood-plates can be seen as nearly or quite colourless bodies.

Under these circumstances it would appear as if all the phosphorus having left the red corpuscles, and all or nearly all the leucocytes and blood-plates, had passed into the plasma, and yet there are no signs of fibrin.

If, instead of allowing the blood to dry slowly at room temperature, it is received on to a slide heated to about 70° C., so that it dries immediately, the corpuscles are killed before they can part with their nucleo-proteid, and a picture is obtained after treatment by the Macallum test, the reverse of that just described, for now the red corpuscles are tinged faintly green, the leucocytes a deeper green, and the matter in which they lie is colourless. The blood-plates, however, cannot be seen at all.

A few drops of blood from the punctured lobe of an ear are collected in a very small test-tube, allowed to coagulate and left for eighteen or twenty hours; the result is a reddish jelly-like clot floating in a pale red serum. The clot is placed in alcohol, imbedded in paraffin, sections cut and tested for phosphorus.

When examined microscopically it will be found that in the main the appearances are similar to those formed in post-mortem clots. There is a thick rim of homogeneous yellow material which forms the clot proper, and in the centre are the red corpuscles closely set and staining, as in unclotted blood, a pale green with a darker rim. The leucocytes and blood-plates are darker green, and there is no trace of any fibrin. Where the homogeneous

yellow rim comes into contact with the red corpuscles (stained green) there is a zone where a number of pale yellow empty-looking bodies (red corpuscles) are embedded in a yellow matter, and passing the eye from the centre to the periphery these red corpuscles, devoid of phosphorus, are seen to become smaller and smaller until they finally disappear altogether in the yellow homogeneous clot.

The serum from the clot smeared thickly on a slide, dried and tested for phosphorus, almost immediately coloured a bright sage green, and presented microscopically the same appearances as the blood films, only the red corpuscles were less numerous and the ground substance was distinctly darker green.

The inferences I draw from the experiments are :—

(1) That the blood as it drops against the glass tube is excited to shed the nucleo-proteid from the red corpuscles, leucocytes, and blood-plates, but that the latter two perish from the conditions of the experiment, and for some unexplained reason the free nucleo-proteid does not go on to combine with the lime salts and flinogen to form fibrin, but remains in solution in the serum, and therefore this part contains an excess of phosphorus, and stains a much deeper green than usual. The clot is not a vital clot, and contains no fibrin. The red and white cells and blood-plates which do not come into contact with the glass tube probably survive longer, at any rate they do not part with their nucleo-proteid contents.

(2) The clot proper evidently consists of the external rim of homogeneous non-nucleo-proteid material, which it would seem is formed by the destruction of the red corpuscles.

When blood drops are allowed to fall into alcohol, the elements, which to a large extent retain their phosphorus, are probably immediately killed by contact with the alcohol before they can shed their nucleo-proteid, and the microscopical appearances are much the same as those presented by non-clotted blood, but here and there are yellow areas which, although under a low power, appear homogeneous, are seen when more highly magnified to be red corpuscles devoid of phosphorus tightly packed together, or even coalescing ; for often the margins of individual cells are not distinguishable. And very probably if this alcoholic clotting could be more slowly brought about, it would be found that these red corpuscles entirely lose their individuality and come to form the



yellow homogeneous material which is the characteristic clot of non-vital origin.

Some observations with regard to the coagulating influence of alcohol on the blood, in tissues fixed by alcohol strongly support this contention.

If small pieces of cortex or other part of the surface of the brain with the meninges attached are fixed in absolute alcohol and neighbouring pieces in saturated sublimate, followed by graded alcohols, and sections prepared, it will be found that nearly all, perhaps all, of those from the alcohol fixed tissues show in the meningeal vessels and sometimes in the larger vessels of the white matter near the cut surface, clots sticking to one side of the lumen. These stain dark green with Unna's polychrome blue, and by this means are quite undistinguishable from clots which it is suggested have a pathological significance, but when tested for phosphorus they do not stain green, and therefore contain no nucleo-proteid. They have a yellow homogeneous appearance quite similar to that seen in the post-mortem clots, except at their inner margin, where the homogeneity is disturbed by more or less clearly defined red corpuscles, devoid of phosphorus and therefore of a yellow colour which lie embedded in the homogeneous matrix.

As the sections from adjoining parts in sublimate fixed tissue never show these laterally placed clots in the meningeal vessels, and as the clots do not contain phosphorus, it is assumed that they are artificial products caused by the alcohol.

Now that I am referring to sublimate fixed tissues I may state that they give, when tested for phosphorus, the same reaction only not so marked as alcohol fixed tissue, a point which might be of some service if it was desired to test clots for phosphorus in cases where the only fixative used had been sublimate.

The blood-plates in the vessels of the tissues stain as they do in clots, a rather darker green than the leucocytes, which in turn are darker than the red corpuscles. Intra-vascular coagula, whether in the form of fibrin threads or free lying spheres, or lobulated masses, or large masses forming a complete cast of the lumen, in all cases stain a dark green, and are by this means sharply differentiated from the clots produced by alcohol.

*Blood-Plates.*—It would seem from these observations that the blood-plates are, as Lilienfeld stated, of a nucleo-proteid nature,

but further, if we may assume that the depth of green colour with phenyl-hydrazin hydrochloride is proportionate to the amount of phosphorus they contain, then they are relatively richer in nucleo-proteid than either the red corpuscles or the leucocytes, and when the very large number in which they occur is taken into consideration, it becomes in the highest degree probable that of all blood elements they represent collectively the chief carriers of nucleo-proteid.

If this be so, and the present views concerning the process of coagulation are true, it may reasonably be assumed that the share they take in this process is of the greatest importance.

The tendency they show to stick to one another, to the leucocytes, and to any foreign body they may be brought into contact with, has been noticed ever since they were first described. In the coagula described in this paper they occurred in large numbers with an exaggerated tendency to cohere. Instances of the various stages they pass through from a single blood-plate to the large irregular masses where individual forms are lost can be readily found in all preparations.

Now although in the description given of vital coagulation it has been associated with fibrin, it is suggested that during certain pathological conditions the plates may fuse together without the formation of fibrin. Coagulation under these circumstances being the result of a less vigorous process than occurs in freshly-shed blood or with certain fibrinous inflammatory conditions.

#### SUMMARY.

A. By means of the Macallum test for phosphorus it is possible to discriminate between the clotting which is the result of vital action, and that which occurs on the application of alcohol to the tissues, or after the death of the blood cells. In the former case the coagulation consists of a nucleo-proteid substance, which stains green. In the latter two cases it consists of a substance absolutely devoid of phosphorus and therefore not nucleo-proteid, but which would seem to be the result of disintegration of the red corpuscles.

When it becomes a question as to the significance of these nucleo-proteid clots, we have still no sure test which will allow us to differentiate between coagulation occurring in a moribund

or recently dead subject which may have no pathological significance, and that which may occur during active life where it would have a very decided pathological significance.

*B.* These observations also confirm Lilienfeld's statement as to the nucleo-proteid nature of the blood-plates, and tend to show that they play a very important part in the process of coagulation ; probably in certain forms the predominant part.

#### REFERENCES.

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2. Schafer's "Text Book of Physiology," Vol. I. Article "Blood."
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## Abstracts

### ANATOMY.

**ON THE CELLS OF THE SPINAL GANGLIA AND ON THE  
(36) RELATIONSHIP OF THEIR HISTOLOGICAL STRUCTURE  
TO THE AXONAL DISTRIBUTION.** W. B. WARRINGTON  
and F. GRIFFITH, *Brain*, Autumn 1904, p. 297.

DOGIEL and others have shown that in the spinal ganglia there are, besides the usual cell with the T-shaped axon of Ranvier, other varieties whose axons are distributed in different ways. The difference in size of the component cells is one of the most striking features of the ganglia. A thousand cells in each of three regions in the cat, viz., the second cervical, eleventh dorsal and seventh lumbar ganglia, were measured. The second cervical receives fibres from the skin and small muscles; the eleventh dorsal, in addition, receives afferent visceral fibres; the seventh lumbar has no visceral connections, but receives the sensory fibres from the large hamstring muscles. Curves constructed from these measurements show that whilst the cervical and dorsal ganglia show great similarity in the proportion of cells of different sizes, in the lumbar region there are a number of cells of larger size, and we conclude that these cells represent the cells in connection with the sensory organs, the muscle spindle of the large hamstring muscles.

It is known from the works of many, especially Lugaro in Italy, and Orr and Rows in this country, that when stained by the

Nissl method, distinct types of cells can be recognised, and we have endeavoured by experimental section of nerve fibres of different distribution, viz., to viscera (white rami), to muscles and to skin, to study the consequent *reaction à distance* (axonal reaction) which occurs in cells of the corresponding ganglia, and to identify the cells thus altered in terms of Lugaro's classification, which in the main we accept.

It was found in this way that two types, the large clear cell and the obscure cell, undergo chromatolytic change, the smallest cells under  $25\ \mu$  always remaining unchanged, and the cell of large granules, which we propose to call the coarsely granular cell, also being highly resistant, as it only shows chromatolytic change when the section is made just beyond the ganglion, as can readily be done in the cauda equina. Numerous illustrations accompany the paper.

#### SUMMARY OF CONCLUSIONS.

(1) The size of the cells in the ganglia is a function of the diameter of the nerve fibre, and the difference in appearance of the chromophile elements has a dynamical significance. These elements represent material which is used up by the essential trophoplasm, which presides over the nutrition of the nerve fibre.

(2) At the region of the limb plexuses, a number of large cells appear in the ganglia, which are the cells of origin of the fibres to sensory muscle structures.

(3) Lugaro's classification is confirmed in the main.

The cells met with are best described as ( $\alpha$ ) clear cells; ( $\beta$ ) obscure cells; ( $\gamma$ ) coarsely granular cells; ( $\delta$ ) smallest clear cells.

(4) The large clear cell and the obscure cell give origin to the fibres distributed to the skin, viscera and muscles. Of these the largest cells are exclusively connected with fibres from the muscle spindles. Large somatic and large splanchnic fibres have origin from cells of the same type, the large clear cell; small somatic and small splanchnic fibres also from cells of the same type, the obscure cell.

(5) Afferent fibres to the thoracic viscera arise from the first five dorsal ganglia. The number of such fibres in one instance was found to be 136 in the first dorsal, 192 in the second white ramus.

(6) The smallest cells in the ganglion, under  $26\ \mu$  in diameter, whether belonging to the obscure or clear type, are immature cells, and have no fully-developed axon.

(7) The coarsely granular cell is highly resistant to the axonal reaction, and may be a "relay" cell, but sufficient evidence is not forthcoming as to its significance.

(8) The cells in the early embryo have the appearance of the

smallest type described in the text. In the newly-born human subject the types present in the adult can be recognised. The morphological characteristics of the cells of early embryonic life are also met with in the reactive phase.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

**FATIGUE.** Sir W. R. GOWERS, *The Quarterly Review*, October (37) 1904.

IN this article Sir William Gowers sets forth in interesting and lucid style the present state of our knowledge of fatigue as a physiological phenomenon. The works on which he explicitly bases his discussion are *Fatigue*, by A. Mosso, Professor of Physiology in the University of Turin (Translation, Swan, Sonnenschein, 1904); *Weariness*, by Professor Sir Michael Foster, K.C.B. (*Nineteenth Century*, September 1893); and *Remarks on Replies by Teachers to Questions regarding Mental Fatigue*, by F. Galton, F.R.S. (*Journal of the Anthropological Institute*, Vol. xviii., 1889).

Fatigue is a double-sided phenomenon. It is primarily a sensation, and as such is too familiar to require description; but it is a sensation which has a definite external manifestation, namely, the loss of power, whether of brain or of muscle, which shows itself in the fatigued subject. It is this outward manifestation which has lent itself to scientific study; and here Sir William Gowers takes occasion to point out the immense debt which physiology owes to simple mechanics. "If the knowledge gained by its means could be eliminated," he says, "that which remains would be little more than was perceived fifty years ago, except in the domain of the chemistry of life." In the study of fatigue, mechanics has come to the aid of physiology in the form of Professor Mosso's ergograph—an instrument now too well known to students to be described here.

*Muscular Fatigue.*—Analogous to the double aspect of fatigue is the twofold nature of its cause. Experiment has shown that a muscle exhausted by work can be rendered again capable of response to stimulation by simple irrigation. This proves that work gives rise to toxic products which hinder or prevent action. But this is not the only—perhaps not even the most powerful—cause of the diminution of strength. When the muscle fibres are excited by a stimulus, the atoms composing them are enabled to enter into closer combinations, and in so doing they bring about the muscular contraction. When the muscle through overwork ceases to be able

to contract, it is due to the exhaustion of those elements from which the energy is in this way derived.

It is to the first of these causes that the sensation of fatigue is thought to be mainly due, and of recent years histology has enabled us to form some definite conception of its place of origin. Each of the sensory nerves which enter the muscle terminates in a peculiar long tapering enclosure known as a muscle spindle; into this passes a contractile muscle fibre which divides inside, and round which the ends of the nerve fibrils are arranged. The condition of the muscular fibre enclosed being conveyed to the brain by the nerve, is taken as an indication of the state of the muscle as a whole, the particular fibre serving simply as a sample. When this fibre contracts, it, like the rest of the muscle, produces toxic substances, and their presence acting on the nerve probably gives rise to the sensation which we call fatigue. It is, moreover, probable that it is more difficult for those products to escape from within the spindle than from the free muscle fibres; and if this is so, it would account for the fact that the feeling of fatigue often persists after the muscle is quite able again to resume work.

*Cerebral Fatigue.*—The facts here, owing to the difficulty of measuring mental exertion, are even more obscure than is the case with the muscles. It is, however, obvious that all muscular work must be accompanied by brain work, sometimes, as for instance in school games, to a very considerable degree; hence it follows that from muscular exertion brain fatigue may directly result. Again, the injurious substances produced in the working muscle pass into the blood, and so very easily reach all parts of the body, including the brain; and to these noxious agents the nervous substance of the brain is even more sensitive than is the muscular tissue. Prolonged muscular work appears to diminish or even destroy the sensory power of the centres; thus, some Alpine climbers require to note down the incidents of their ascent as they occur, for they find their memory quite unable to retain them.

In brain work proper we find the same two causes acting to produce fatigue as in muscular exertion, viz., the harmful products of activity and the exhaustion of the tissue power. By experiments with the ergograph, Professor Mosso has demonstrated that cerebral exhaustion is accompanied by a very marked decrease of muscular power. This, Sir William Gowers thinks, "can only be ascribed to a lessened degree of the nerve energy which stimulates the muscles." But Professor Mosso expressly states that several of his remarkable tracings taken before and after mental work were written, not by voluntary, but by involuntary movements of the finger—the electrodes being applied directly to the muscles of the forearm. Hence he draws the conclusion that the diminution of power shown in the tracings does not proceed merely from

the spread of the effects of fatigue in the brain, but that the cerebral exertion has actually brought about the deterioration of the muscular tissue itself.

The other results of brain fatigue, however, are both more important and more generally known. The "mental grasp" is usually lessened, and mental work is accompanied by more sense of effort; often, too, the power of recollection is weakened. But the results here are of such extreme variety that it is difficult to make generalisations. Many of the signs of fatigue collected by Galton arise from individual idiosyncrasies; some, such as the tendency of the letters of print to run together, can, as Sir William Gowers points out, be traced to some particular weakness in the subject—in this special instance to inherent weakness in the muscle of the eye which adjusts the focus—the tendency of any strain being, of course, to affect the weakest spot first. One of the most common results of brain fatigue is headache, the precise origin of which is still a mystery to science.

It is often maintained that fatigue is prevented by change of work. Sir William Gowers deals with this question in the light of our present physiological knowledge, and finds that it has a very considerable substratum of truth. The nerve elements apparently know not rest; they are ceaselessly in a state of gentle activity, an activity which entails a constant change of the atoms which compose them. The increase of these nutritional changes by moderate work will bring about greater vital efficiency, and change of mental work may conduce to the gentle activity which is most favourable to this rebuilding of the cerebral substance. Again, any brain work increases the amount of blood supplied to the brain as a whole; but, as the different parts of the brain are involved to very different degrees in the various forms of brain activity, a change of work may leave the exhausted elements almost at rest, and yet, by an increased flow of blood, facilitate the removal of their waste products and the assimilation of the new elements of which they are in need. Thus, in these two ways, change of mental activity may promote the well-being of the brain. Yet it must not be supposed that a mere alteration of work is recreative in any true sense of the word. To rid the organism of the harmful effects of fatigue, rest and recreation are both indispensable, but the recreation, to be worthy of the name, must involve an entire change in the character of the nerve activity. Many a holiday is rendered valueless by neglect of this simple common-sense dictum.

MARGARET DRUMMOND.

**PSYCHOLOGY.****ON PANPSYCHISM AS AN EXPLANATION OF THE RELATION-**

(38) **SHIP BETWEEN MIND AND BODY.** (*Sur le Panpsychisme comme explication des Rapports de l'Ame et du Corps.*)

M. TH. FLOURNOY, *Arch. de Psychol.*, S. iv., No. 14, p. 129.

**SOME CONSIDERATIONS ON PANPSYCHISM.** (*Quelques con-*

(39) *siderations sur le Panpsychisme.*) Prof. C. A. STRONG,

*Arch. de Psychol.*, S. iv., No. 14, p. 145.

BOTH of these papers were read at the Second International Congress of Philosophy at Geneva in September last, the second paper being a reply to the first. The various problems which are discussed appear to belong rather to the domain of epistemology than to that of psychology.

The doctrines of panpsychism are not new, but they have been lying dormant, and Professor Strong, who has proved a most able exponent, has recently given them a fresh stimulus and impetus in his remarkable book, entitled "Why the Mind has a Body."

The main doctrine of panpsychism is, according to Professor Strong, that consciousness is the only reality. Matter is not a reality, but a phenomenon; it is a symbol of things in themselves, whereas mental states are not symbols. And, since consciousness is the only reality of which we have any immediate knowledge, and therefore the only sample of what reality is like, we have no other conception of a reality. Hence Professor Strong concludes things-in-themselves to be purely mental in nature. More specifically, the brain of others is only the mode or symbol by which the consciousness of others appears to me.

The first of these two papers is a criticism of panpsychism as expounded in Professor Strong's book. M. Flournoy tells us that, of all the prevailing theories of the relationship between mind and matter, panpsychism attracts him most; but, at the same time, he points out various difficulties which prevent him from freely accepting it. Professor Strong's book is an attempt to do away with psycho-physical dualism; but, as M. Flournoy says, the difficulties in the way of accepting the latter doctrine are merely displaced, they are not removed. If the statement is made that the brain of others is only the mode in which I perceive their consciousness, this does not remove the dualism between the consciousness of others and the representation I experience in the symbol of a material brain.

The answers to M. Flournoy's various objections are clearly expressed, but one finds oneself wondering whether Professor



Strong really means what we understand him to mean. To one who is accustomed to think in terms of psycho-physical parallelism, everything appears to be turned upside down. Whereas we have been accustomed to regard a percept as the symbol of a "thing-in-itself," we are now told to regard a "thing-in-itself" as the symbol of a percept. There are many hard sayings in both these papers, and it is difficult to do them justice in an abstract.

W. H. B. STODDART.

### CLINICAL NEUROLOGY.

**THE SEVENTH CERVICAL RIB AND ITS EFFECTS ON THE**  
(40) **BRACHIAL PLEXUS.** WILLIAM THORBURN, F.R.C.S., *Trans.*  
*Roy. Med. and Chir. Soc. of London*, 1904.

IN this important paper Mr Thorburn rightly points out that the presence of a 7th cervical rib has been insufficiently considered by Neurologists. Its unsuspected presence may be the cause of symptoms associated with a lesion of the lower trunk of the brachial plexus, and, as the author has demonstrated, the morbid condition may be relieved by operative measures directed to the removal of the rib. The early symptoms are: (a) pain radiating from the neck down the inner side of the forearm; and (b) cramps and vasomotor changes in the fingers. Later appear atrophic palsy of the muscles supplied by the 8th cervical and 1st dorsal roots, and blunting of sensation in the same distribution. Local signs are sometimes detected in the form of increased pulsation of the sub-clavian artery under the examining finger, and a prominence due to the presence of the rib in the posterior triangle of the neck. It is noteworthy that, whereas these accessory ribs are present in a certain percentage of persons of both sexes, they tend to produce symptoms chiefly in women, and nearly always on the right side. The explanation of this predisposition is not explained. The differentiation of these cases from those of uniradicular palsy can be made by excluding the presence of the rib, and also by the fact that in the latter class, when the lesion is situated in the 1st dorsal root, the sensory loss is sharply limited to an area extending from the elbow to the ulnar side of the wrist, and is dissociative in character.

E. FARQUHAR BUZZARD.

**HETEROTOPISCHE INNERVATION.** W. G. HUET, *Neurolog.*  
(41) *Centralbl.*, 1904, p. 1085.

IN an otherwise healthy man, who was complaining of some minor rheumatic affection of the left shoulder, it was observed on inspec-

tion of the back, that, every time he spoke, a small bundle of fibres in the right trapezius muscle was thrown into contraction. This bundle took its origin from the spines of the third to the fifth dorsal vertebræ, and ran obliquely downwards and outwards to be inserted into the spine of the scapula. Its breadth was about 5 cm. It stood out from the rest of the muscle whenever the patient phonated, not during whispering. Less marked contraction was also palpable, though scarcely visible, during swallowing. No alteration in the position of the scapula was thereby produced. The ordinary voluntary movements of the whole trapezius were normal, also its electrical reactions. Laryngoscopic examination showed no abnormality of the cords. The left trapezius was normal.

Huet discusses various possible explanations of this remarkable phenomenon. The condition is analogous to the contraction produced by stimulation of the hypoglossal nerve after hypoglossofacial anastomosis, in which—the distal end of the hypoglossal having been united to the proximal end of the facial—innervation of the face causes associated movement of the corresponding half of the tongue. He recalls cases of accessorius palsy associated with palsy of the soft palate, pharynx and larynx (but these are probably due to implication of the adjacent roots of the vagus).

In this particular case he inclines to the idea that the vagus nucleus innervated the larynx, *via* certain strands of the accessorius. This opens the interesting question of the original connection between muscle and nerve—whether, as Gegenbauer maintains, motor nerve and muscle-fibre are in a primal unalterable connection, or whether, as His believes, the nerve-fibre starts from the central nervous system and grows out towards its corresponding muscle-fibre. Huet's case would be an example of the fibres having, so to speak, lost their way and gone round *via* the accessorius. Finally, there are developmental facts which bear on the question. The shoulder-girdle, according to Gegenbauer and Fürbringer, is originally developed from the hindmost branchial arch. And in the skate the anterior part of the trapezius is inserted into the last branchial arch, whilst the posterior part forms a special muscle, the *constrictor superficialis dorsalis*. The nerves of these two muscles are derived from the most posterior portion of the vago-accessorius nucleus.

PURVES STEWART.

#### **THE SENSORY DISTRIBUTION OF THE FIFTH CRANIAL (42) NERVE.** *Johns Hopkins Hospital Bulletin*, Vol. xv., 1904.

THIS paper presents the results of the investigation in 26 cases of the sensory loss which follows extirpation of the Gasserian ganglion, and is supplemented by the study of the anæsthesia

which follows destruction of the upper cervical nerves, or after removal of their dorsal root ganglia.

Cushing's conclusions on the outline of the trigeminal cutaneous field are more or less in agreement with the views of most clinicians, but the definition of its borders and of its characters is more definite than any hitherto published descriptions.

The boundary of complete anæsthesia, starting from the middle line above, passes downwards approximately over the Rolandic fissure, "then drops to the anterior attachment of the pinna, around the edge of which it curves in a backward direction, so as to include a small section of the ascending rim of the helix, together with the entire crus of the same; thence it disappears in the external auditory canal at the upper edge of the meatus; from this point the line passes into the canal along its upper wall as far as the tympanic membrane, which is included to a greater or less extent in the anæsthetic area, returns along the lower and anterior wall of the canal to the lower edge of the tragus, where it once more reappears on the exposed cutaneous surface; thence it passes at a greater or less angle in a forward and slightly upward direction across the zygomatic region for a distance of from 3 to 5 cm. before turning and sweeping downward across the cheek, still keeping nearly 5 cm. from the posterior edge of the ascending maxillary ramus."

In rare cases (3 out of 26) this limiting line was set further back, so as to enclose a greater portion of the cheek, the upper half of the pinna and the concha.

Though within the area so limited there is complete tactile loss, it includes a strip limited posteriorly by this boundary line, over which painful stimuli are perceived, but only as touch. This strip is very narrow on the cheek, somewhat wider in front of the tragus and in the scalp.

The medial limit of the anæsthesia corresponds exactly to the middle line of the skull and face.

Within the mouth the anæsthesia is limited by the mid line of the lip, frænum linguæ, and tongue as far back as the foramen cæcum, whence it passes lateralwards along the circumvallate papillæ to the anterior pillar of the fauces, and along it to the uvula. It is doubtful if the anæsthesia of the nasopharynx is continuous with that of the mouth.

Though this oral anæsthesia is complete in the ordinary tests, it is generally found that some sensation results if anything be drawn over the tongue. From this Cushing assumes that, besides the taste fibres which the chorda tympani and nervus intermedius carry, these nerves also contain fibres for common sensation from the anterior two-thirds of the tongue.

There is remarkably little sensory overlapping between the

trigeminal and spinal nerves, and thereon is probably dependent the remarkable permanence of the anæsthesia.

Some flattening and flaccidity of the same side of the face and asymmetry of the palate, which may result from section of the trigeminus, is probably attributable to loss of muscle-sense.

No definite statement can be made about the sensory supply of the dura mater, but the trigeminus probably takes considerable part in it. The observation that headache from any cause is generally limited to the opposite side after section of the trigeminus, is of special interest.

The paper concludes with an interesting discussion of the development of the trigeminal supply. In the embryo this nerve supplies the head in front of the first visceral cleft and the anterior wall of the latter, from which the facial side of the external auditory meatus, the tragus, and part of the helix develop, *i.e.* those portions which later derive their sensory fibres from the trigeminus.

GORDON HOLMES.

**ON SO-CALLED FACIAL HEMIHYPERTROPHY.** H. MACKAY,  
(43) *Brain*, Part iii., 1904, p. 388.

THE first part of this paper consists of a critical digest of the literature of hemihypertrophy, followed by abstracts of many of the reported cases. Mackay's own case is that of a girl of ten suffering from acquired hypertrophy of the left side of the face and skull. It seems clear that the abnormality was not congenital (as in most instances), because in photographs taken when the patient was five years old there is no asymmetry. The overgrowth began as a bony swelling near the vertex, spreading to the occiput, forehead, and finally to the cheek. The parts affected at the time of examination were the whole of the soft parts of the left side of the face, all the left facial and most of the cranial bones—the frontal, parietal, sphenoidal, temporal, and occipital. There was marked bossing of the frontal, parietal, and occipital eminences. The malar, zygoma, and mastoid were enlarged, and the molars longer and thicker on the left side. The vision of the right eye was normal, that of the left  $\frac{6}{36}$ , not improved by glasses, the visual field being contracted on the nasal side. Hearing was normal on the right side, defective on the left. No vasomotor or vascular anomalies were present; there was hypertrichosis on the left cheek. After describing the case, Mackay gives a digest of the clinical features of the condition, but this, based as it is on a number of published cases, it is not possible to abstract here. He is of opinion that it is impossible, and, on pathological grounds, of doubtful validity, to separate congenital from acquired cases. As

hereditary transmission is unknown, the condition is, in a biological sense, always acquired. In his short reference to the pathology, a recent case published by Robert Hutchison in the "Transactions of the Society for the Study of Diseases of Children" has escaped notice; in it a full histological examination of the tissues was made, and the hemihypertrophy was found to extend to the paired viscera. The theories of the pathogenesis are passed under review—that of vasomotor paresis, which finds favour with so many writers, that of circulatory stasis produced by vicious positions *in utero*, that of stimulus or irritation of epiphyseal cartilage and osteoclastic periosteum, the purely teratological explanation, which, however, fails to explain acquired cases, and the tropho-neurotic theory. To the last of these he leans, finding support in the occasional occurrence of hypertrophic dystrophies in syringomyelia and acromegaly. He states that, except in his case, occipital bossing has never been described in hemihypertrophy, and suggests that it points to a possible connection with acromegaly. Cases of hemihypertrophy affecting the soft parts alone are probably allied to Herloghe and Rapin's facial trophoedema. He concludes that: (1) While nothing is known of the pathogeny of facial hemihypertrophy, the most reasonable explanation of the condition is that which associates it with disturbance of hypothetical nutritional or trophic centres in the periependymal grey matter of the encephalon. (2) In some of its features, facial hemihypertrophy appears to have relationship with syringomyelia and with acromegaly. A full bibliography is appended to the paper.

J. S. FOWLER.

#### CLINICAL OBSERVATIONS ON DIPHTHERITIC PARALYSIS.

(44) J. D. ROLLESTON, *Practitioner*, Nov. and Dec. 1904.

#### DIPHTHERITIC PARALYSIS CURED BY SERUM. (Paralysies

(45) *diphtériques guéries par le serum.*) COMBY, *Archiv. de Méd. des Enfants*, July 1904, p. 411.

ROLLESTON (*Practitioner*, Nov. and Dec. 1904) writes on Post-Diphtheritic Paralysis, with notes of its incidence in a series of 500 consecutive cases of diphtheria. He points out that the apparent increase of this condition is due, firstly, to the fact that serum treatment saves a large number of severe cases which in other times would have died, without having had time to develop this sequela; and secondly, to the more accurate observation which increased interest in diphtheria has undoubtedly caused. As regards the occurrence of paralysis in its relation to the severity

of the initial attack, he takes the view that it is undoubtedly more frequent after severe attacks. This view is most strikingly supported by his tables of cases. Rolleston points out that the occurrence of paralysis after a mild attack is probably often due to the patient being allowed to exert himself too soon. As regards laryngeal cases, he agrees with most authorities that paralysis is rare unless there is a faucial complication also. Curiously enough, he does not draw attention to the frequency of paralysis after cases where both fauces and nose are affected. Of 150 consecutive cases of paralysis, in the reviewer's experience, no less than 47, or practically one-third, had had a nasal lesion; and putting it in another way, of 116 cases with a nasal lesion, no less than 47, or not very far from half, subsequently suffered from paralysis. This is a very much higher percentage than ever follows faucial cases alone. As regards age, adults are relatively immune from paralysis. Rolleston gives an interesting table showing the percentage of each form of paralysis in his cases. As usual, palatal palsy is much the most frequent, and is followed by ciliary paralysis in order of frequency. Strabismus is considerably less common. Here, again, it is curious that no mention is made in this table of paresis of the lower extremities, which is surely a definite and specific enough form of weakness to be included. Granted that this paraplegia is often difficult to distinguish from the weakness resulting from the disease and confinement to bed, it is nevertheless a very real complication in many cases. An interesting table is given showing the time of onset of the various forms of palsy, and another showing the average duration of each form. As regards the interesting cardiac conditions so frequently causing death, Rolleston lays great stress on the increase of size of the liver, which in some instances he found below the umbilicus. In the treatment of heart failure he thinks strychnine useless, and believes adrenalin administered freely may frequently save life. This is a most valuable suggestion, and well worth an extended trial, as there is no condition which is a greater opprobrium to modern therapeutics.

Comby (*Archiv. de Méd. des Enfants*, July 1904) reports a few cases of post-diphtheritic paralysis treated by antitoxin. In every case the injections were followed by marked improvement. But in the first place it is difficult to see how serum is going to improve a lesion already existing, and in the second a perusal of Rolleston's statistics, in the paper noted above, illustrates admirably the abortive character and short duration of the majority of diphtheritic palsies. This characteristic will always make it most difficult to attribute rapid improvement to any particular mode of treatment, and we would require reports of a very large series of cases before admitting that Comby has proved his point.

CLAUDE B. KER.

**THE RELATIONS BETWEEN THE TOPOGRAPHY OF THE**  
**(46) PARALYSES AND OF THE ALTERATIONS IN THE**  
**MOTOR CENTRES IN ACUTE ANTERIOR POLIOMY-**  
**ELITIS OF CHILDHOOD.** (Sur les rapports entre la topo-  
 graphie des paralysies et celle des altérations des centres  
 moteurs dans la poliomyélite antérieure aiguë de l'enfance.)  
 PARHON et PAPINIAN, *Journ. du Neurol.*, Nov. 22, 1904, p.  
 421.

THIS paper presents the pathological study of the case of a woman deceased when eighty-two years of age, who in infancy had an attack of acute poliomyelitis affecting the one leg. This limb was as a result ill-developed, and there was complete paralysis and extreme wasting of all the muscles of the foot and leg, and of the quadriceps extensor, the adductor magnus, and the biceps cruralis. Other muscles of the hip and thigh were affected in slighter degree. On microscopical examination it was found that the wasted muscles were in large part replaced by adipose tissue, with here and there the altered remains of muscle fibres. These stained very poorly, were generally granular, their transverse situation often lost, with often a tendency to longitudinal striation or splitting. There was great proliferation of the nuclei, increase of fibrous tissue, and the muscular coats of the vessels were uniformly hypertrophied.

Some atrophy of the upper end of the ascending frontal convolution was observed, but the cortex appeared normal on Nissl staining.

The lower four lumbar, and the upper two sacral anterior roots were atrophied, but no change could be detected in the spinal ganglia. Various groups of anterior cornual cells were absent, and here and there the remains of altered vessels were observed in their place, which the authors assume indicates the vascular origin of the pathological process.

They describe various muscles as deriving their nerve supply from groups of anterior cornual cells:—

*Leg muscles*—the postero-lateral group which extends from the lower portion of the 4 L. to the 3 S. segments.

*Foot muscles*—from the post-postero-lateral group of the upper three sacral segments.

*Biceps cruralis*—from the central group of 1 S. and upper part of 2 S. segments.

*Semi-membranosus and Semi-tendinosus*—from the central group above 1 S. segment.

*Sartorius*—antero-lateral group 3 L.

*Quadriceps*—lateral group 3 L.

*Gracilis and Adductors*—central group 3 L.

*Glutei*—antero-lateral group 5 L. and upper two sacral segments.

There is a needless discussion as to whether the distribution of the paralysis in poliomyelitis is of the root type, *i.e.* if the muscles affected depend on the roots from which they receive their nerve supply, but they rightly negative this idea. GORDON HOLMES.

**THE ARRIS AND GALE LECTURES ON THE NEUROLOGY OF  
(47) VISION.** By J. HERBERT PARSONS, 1904.

THE subject matter of this interesting and valuable brochure is arranged under two headings: Lecture 1, The Afferent Visual Paths; and Lecture 2, The Innervation of the Pupil. In a comparison of the anatomical structures for the transmission of visual and common sensations, the writer considers that the neurones of the first order, in the retina, are the rod and cone bipolars rather than the rods and cones. The rods and cones will then be epithelial ependymal cells.

The neurones of the second order are the ganglion cells of the retina and their processes. Their axones pass into the nerve fibre layer of the retina, thence into the optic nerve and chiasma, where most of them cross to the optic tract. From the optic tracts most are distributed to the external geniculate bodies, but others pass to the superior colliculi, and yet others to the pulvinar of each optic thalamus.

Incorporated in this lecture is a description of the writer's experimental researches upon monkeys on the arrangement of the optic nerve fibres (see *Brain*, 1902).

The grey matter of the lateral geniculate body contains the cell bodies and dendrites of the optic neurones of the third order. Cells in the pulvinar around which fibres from the optic tract arborise, and ganglion cells of the superior colliculus are also optic neurones of the third order.

The fibres from the lateral geniculate body to the cortex cerebri pass to Wernicke's field, an area situated on the outer side of the external geniculate body. Here they are joined by similar fibres from the superior colliculus and pulvinar, the latter being ventral to those derived from the geniculate body. They all turn round the caudate nucleus and enter the optic radiation of Gratiolet. The fibres from the lateral geniculate body tend towards the neighbourhood of the calcarine fissure. In comparing the nuclei under consideration with the afferent tracts of sensibility, the ganglion cell layer is found to correspond with the nuclei gracilis and cuneatus, whilst the termination of the optic tract in the superior colliculus and thalamus (lateral geniculate



body and pulvinar) corresponds with the terminations of the nucleo-thalamic (mesial fillet) tract in the mesencephalon and diencephalon respectively. An account of the cerebral visual centres occupies the remainder of Lecture 1.

Lecture 2 contains an historical resumé of the literature upon the subject of the innervation of the pupil, and the conclusions arrived at concerning the course of the pupil-constrictor fibres and the dilator tract as the result of a vast amount of research during the latter part of last century. From histological, taken in conjunction with physiological evidence, it must be now considered proved that there is a dilator muscle in the iris.

Paradoxical pupillo-constriction and paradoxical pupillo-dilatation are subjects dealt with at considerable length. The former is thought by Anderson to be due to increased excitability of the denervated sphincter, brought about, in certain observations, by alterations in the blood supply, eserine, and probably ether.

The writer uses the term synkinesis for an associated movement of the pupil. There are two chief reflexes and two chief synkineses—the light reflex, the accommodation synkinesis, the sensory reflex, and the cerebral synkinesis.

As regards the afferent fibres concerned in the light reflex, it is still conjectural by what path they pass from the optic tract to the third nerve nucleus.

The accommodation synkinesis is a constrictor effect commonly associated with accommodation. It is more nearly related, however, to convergence.

The sensory reflex is a complex effect. There is a primary rapid dilatation, due to augmentation of the dilator tone through the cervical sympathetic, followed by a second dilatation, rapid in onset, but very slow in its disappearance, due to inhibition of the constrictor tone.

The cerebral synkinesis is a complex phenomenon. It is not a simple reflex. It is induced by psychic stimuli. It is also largely an associated movement accompanying eye movements which may be themselves due to sensory impulses.

The author's own observations on the relationship of the cerebral cortex to the movements of the pupil (see *Journal of Physiology*, vol. 26) are discussed in this work. A full bibliography is appended.

C. H. USHER.

**THE QUESTION OF CHOKED DISC.** (Zur Frage der Stauungs-(48) pupille.) By Professor UHTHOFF, Breslau, *Neurol. Centralbl.*, October 1904, S. 930.

THIS paper gives an analysis of the cases which had come under Professor Uhthoff's observation during a long term of years. In

a recent publication Dr Kampherstein has given an account of the microscopical examination of 51 eyes derived largely from the same source. Professor Uhthoff's cases number 204, and are grouped by him as follows :—

Brain tumour . . .	in 134 cases.
Cerebral syphilis . . .	„ 27 „
Cerebral tubercle . . .	„ 9 „
Brain Abscess . . .	„ 7 „
Hydrocephalus . . .	„ 7 „
Meningitis . . .	„ 2 „
Cysticercus cerebri . . .	„ 2 „
Sinus thrombosis . . .	„ 1 „
Bony cicatrix on skull . . .	„ 1 „
Malformation of skull . . .	„ 3 „
Nephritis . . .	„ 3 „
Nephritis with lead poisoning „	1 „
Anæmia . . .	„ 2 „
Uncertain diagnosis . . .	„ 4 „

The preponderance of brain tumour over other causes of choked disc is strikingly evident in the series. According to Uhthoff, a choked disc in cerebral syphilis is usually an indication of the formation of a true gummatous mass in the brain. A gummatous basal meningitis is a rarer cause. In tubercular affections of the brain, as is well known, it is the solitary tubercle which has a marked tendency to be associated with choked disc. Tubercular meningitis very seldom gives a typical choked disc, though slight optic neuritis is oftener observed. In hydrocephalus, simple atrophy is more frequent than choked disc, and results from pressure due to dilatation of the third ventricle. The occurrence of choked disc with nephritis is considered by Uhthoff to be very rare.

The condition of pseudo-neuritis optica, a congenital anomaly where the ophthalmoscopic picture of choked disc may be so closely simulated as to deceive an expert, should be borne in mind, lest a too hasty judgment lead to a grave error of diagnosis.

Professor Uhthoff thinks that a prominence of at least 2 droptres ( $\frac{2}{3}$  mm.) is necessary before the condition can fairly be regarded as choked disc. In discussing the etiology, stress is laid on increased intra-cranial tension as an important factor, but the subject is not discussed in any detail.

J. V. PATERSON.

**THE PAINS OF TABES.** Sir WILLIAM R. GOWERS. *Brit. Med. (49) Journ.*, Jan. 1905, p. 1.

IN this clinical lecture the author analyses the characters of tabetic pains in his usual clear and critical manner. They may be classified into the following divisions:—*A.* Those pains which are brief and of momentary duration, but which succeed each other at short intervals and occur in the same area, often for hours, sometimes for days. These brief pains can be grouped into two important sub-divisions. 1st, The superficial, which seem to be just on or beneath the surface, and are usually felt at one spot over a smaller or larger area. They occur most frequently in the legs and feet, and down the ulnar border of the forearms. These are the characteristic "lightning pains," and when frequent, often produce extreme tenderness of the skin to tactile impressions, while pain stimuli are not felt at all, a fact which suggests that these superficial pains may be due to a morbid process in the tactile nerves. 2nd, The deep brief pains which are often ill-defined as to their site and generally last for longer than the superficial pains. *B.* The second great division of tabetic pains includes those which are prolonged—lasting for some time, and varying considerably in their character and intensity. They occur more frequently on the trunk than elsewhere. The best examples of this kind of pain are usually deep, though the superficial girdle sensation is a noteworthy exception. The second type of fixed pains consists of widely diffused and different forms of paræsthesia, such as numbness, tingling sensations of heat and cold, etc.

After reviewing the pains of ordinary tabes, the author describes, under the name of Tabetic Neuralgia, a definite type of case, of which he has seen several examples. The chief features are the presence of severe pains of varying character, the absence of ataxy, and the retention of the knee-jerks, while the pupillary changes may be slight. In one such case there was optic atrophy. As regards the source of tabetic pains, he inclines to the opinion that they arise from morbid changes in the extremities of the peripheral nerves.

The treatment of such pains varies—the superficial may be relieved by chloroform sprinkled on lint with oiled silk over it, or by applying the positive pole of a voltaic battery soaked in a 6-10 per cent. solution of cocaine, which abolishes the sensibility to touch and pain in a few minutes, and affords speedy relief. Amongst the internal remedies, the coal tar products are the best, while in some cases chloride of aluminium in 5-10 gr. doses would seem to diminish the tendency to pain, and lessen both its frequency and intensity.

T. GRAINGER STEWART.

**A CASE OF JUVENILE TABES IN A FAMILY OF NEURO-  
(50) SYPHILITICS.** (Father syphilitic, mother tabetic, brother  
paretic, sister hemiplegic.) J. GRINKER, *Journ. of Nerv. and  
Ment. Dis.*, Dec. 1904, p. 753.

THIS is a careful record of an interesting case of juvenile tabes and of the nervous affections of other members of the family.

The father of the patient had suffered from syphilis. The mother had developed syphilitic symptoms soon after her marriage. At a later date, symptoms of tabes developed. These were shooting pains, optic atrophy, loss of knee-jerks and of the tendo Achilles reflexes, ataxia, Rhomberg's symptom, gastric crises, impaired tactile sensation in the region of distribution of the ulnar nerves, analgesia in the distribution of the ulnar and peroneal nerves, slight trunk anæsthesia, Biernacki's sign (analgesia of the ulnar nerve trunk on pressure), cushion-like feeling under the feet.

The autopsy revealed the changes of advanced tabes in the spinal cord.

A son of the woman, whose symptoms have just been recorded, developed a mental affection, and died from general paralysis of the insane. A sister of last patient (daughter of the woman whose case is first reported) suffered from hemiplegia and Jacksonian epileptic attacks at the age of 17. Signs of hereditary syphilis had also developed—interstitial keratitis, disseminated choroiditis, condylomata, chronic oozena, destruction of the hard palate, snuffles, etc.

The man, whose tabetic symptoms are reported in detail, was the brother of the two patients just mentioned (the son of the woman whose tabetic symptoms are first described). He was 25 years of age when he came under observation. At the age of 3 months he had "moist papules around the anal margin, and rhagades about the oral ring." At the age of 4 months he had a generalised eruption. Six years before he came under observation, *i.e.* at the age of 19, he discovered that the right eye was quite blind. Shortly afterwards, vision began to fail in the left eye. For 18 months he had been quite blind. About the age of 20 he had commenced to suffer from shooting pains in the legs. Afterwards the legs became numb, and a girdle sensation developed.

Ophthalmoscopic examination revealed double optic atrophy. The pupils were dilated, and did not react either to light or accommodation. There was drooping of the left upper eyelid and paresis of the left internal rectus. Other symptoms were: "ataxia of station and of motion, loss of the deep reflexes." Bone sensibility to the vibrating tuning-fork was greatly diminished.

From a perusal of the literature of juvenile tabes, Grinker concludes that "optic atrophy, vesical trouble, and sensory disturbances are among the early symptoms of juvenile tabes, and that syphilis has invariably been present in the antecedents of the patients."

The diagnosis of this case, and the possibility of a form of syphilis having a special tendency to affect the nervous system, are carefully discussed.

R. T. WILLIAMSON.

**AN ADDITIONAL CASE OF PRECOXIOUS TABES.** J. GRINKER, (51) *Journ. of Nerv. and Ment. Dis.*, Dec. 1904, p. 773.

At the time of examination the patient was 50 years of age, but his symptoms had commenced at the age of 22. The first symptoms had been shooting pains in the legs and arms. When examined in 1904 the chief symptoms were Argyll-Robertson pupils, Rhomberg's sign, ataxia of the legs and arms, ulnar analgesia, a broad zone of trunk anæsthesia, absence of deep reflexes, girdle sensation.

R. T. WILLIAMSON.

**THE PROLONGED GASTRIC CRISES OF TABETIC MORPHINOMANIA.** (52) **MANIACS.** (*Crises gastriques prolongées des tabétiques morphinomanes.*) A. BRAUER and DOBROVITCH, *Revue Neurol.*, Dec. 15, 1904, p. 1153.

Two cases are reported in which tabetic gastric crises assumed a peculiar and characteristic nature under the influence of morphinomania. In both cases the crises were observed before the establishment of the morphine habit.

The early effect of the morphinomania was to shorten the intervals between the occurrence of the crises, the latter being at first less severe than usual. Precursory symptoms of the crises appeared, such as vague pains, nausea, or general shooting pain.

The individual crises lasted longer and longer, and the intervals between them became progressively shorter, until a state of continual crises was developed. The vomiting tended to be incessant, and was associated with great effort and little result. Oliguria was associated as in ordinary gastric crises.

Before this condition was assumed, it was easy to distinguish pseudo-crises which occurred for the purpose of satisfying the morphia craving. These pseudo-crises were very like real crises, with the important difference that they were not accompanied by oliguria. They were rather more dramatic than real crises, and

left the mind of the patient freer to act—his attention could be attracted from his suffering, and they tended to occur on occasions where they could attract more attention.

The authors regard these pseudo-crises as combinations of the gastric crises of the morphinomania with tabetic crises.

The gradual demorphinisation of these patients resulted in the crises returning to their original type and interval. The resumption of the habit on leaving hospital caused the pseudo-crises, and later the continual crises to return, and again demorphinisation produced a return to the original state.

The authors point out the urgent necessity of avoiding the use of morphia in the treatment of gastric crises in tabes, on account of the readiness with which some tabetics acquire the morphia habit with the ultimate result, that the crises become continuous. In the treatment of the pseudo-crises of the tabetic morphinomaniac, they consider that strict isolation of the patient is most important.

JAMES COLLIER.

#### **PROGNOSIS OF ACUTE HÆMORRHAGIC POLIO-ENCEPHALITIS**

(53) **SUPERIOR (WERNICKE).** (Ueber die Prognose der akuten hæmorrhagischen Polio-encephalitis superior.) W. SPIELMEYER, *Centralbl. f. Nervenhk. und Psychiat.*, Nov. 1904, p. 673.

WHILE admitting that there are transitional forms of disease connecting acute hæmorrhagic polio-encephalitis superior with other varieties of encephalitis, Spielmeyer believes that the former is a distinct entity on the grounds of ætiology, symptoms and clinical course, as well as of pathological anatomy.

He finds in literature 18 (possibly 20) cases of this condition which ended fatally, and in which the diagnosis was confirmed by autopsy. He gives notes, clinical and pathological, of an original case. There are thus 19 (possibly 21) cases in all.

Clinically, all these cases have three cardinal groups of symptoms: (1) ocular paralysis of acute onset; (2) grave affection of the sensorium; (3) ataxic phenomena, especially in gait and speech.

Ætiologically, the great factor is chronic alcoholism—it was present in all the cases except one.

Pathological changes are found in the grey matter of the aqueduct of Sylvius; punctiform hæmorrhages with granular cells in their neighbourhood, markedly dilated arterioles and capillaries, new formation of vessels, little change in the vessel walls. In contrast to what is found in other forms of encephalitis, there is in the vast majority of cases no sign of true inflammation, notably infiltration of leucocytes.

As regards pathogenesis, changes in the vessel walls are very rare and can play no important rôle. Spielmeyer finds many analogies between the occurrence of these hæmorrhages in the central grey matter and the extravasations which are seen in the hæmorrhagic diathesis; the common cause of polio-encephalitis, viz., chronic alcoholism, predisposes to extravasations of blood in the central nervous system. A still more important factor in the pathogenesis is the presence of newly formed vessels in the central grey matter, due to chronic alcoholism, this excessive vascularisation of the central grey matter is regularly found in polio-encephalitis.

With regard to non-fatal cases of this condition, Spielmeyer accepts only those which have shown the three cardinal symptoms, including grave affection of the sensorium, delirium or somnolence. Even these must be accepted with reserve, in the absence of pathological confirmation. Of such he finds twelve recorded in literature. In all, with one exception, chronic alcoholism was the great ætiological factor. Only two completely recovered; in the others, the ocular paralysis disappeared more or less completely, while the acute mental symptoms gave place to a condition of chronic delirium.

Are there any points of prognostic significance? The author finds that a comparison of fatal with non-fatal cases shows that little help can be got from local symptoms (*e.g.* the ocular paralysis, in relation to its character, extent or progressiveness). He emphasises the fact that the prognosis in an individual case must depend mainly on the degree of affection of the sensorium and on the general physical condition; a rapidly deepening coma and marked affection of organs by alcoholic abuse are the most certain signs of fatal termination.

In conclusion, he states his belief that there is justification for the view that polio-encephalitis is not a disease *sui generis*, but that it, like Korssakow's psychosis and polyneuritis, is a symptom of a general disease, resulting almost exclusively from alcoholism.

A. W. MACKINTOSH.

**THE PROGNOSIS OF EPILEPSY.** WILLIAM ALDREN TURNER,  
(54) *Edin. Med. Journ.*, Dec. 1904, p. 509.

DR TURNER has analysed 526 cases of epilepsy selected as suitable for statistical purposes in relation to prognosis from the records of the National Hospital and the Chalfont Colony. Cases in which there was a "co-existing complication, such as organic cerebral disease," and cases of "idiocy and pronounced imbecility" were not included in the series. The following are some of the con-

clusions arrived at:—"Sex plays little part in the general prognosis of epilepsy. . . . A family tendency to either epilepsy or insanity, though offering no obstacle to the arrest of the seizures in favourable cases, materially increases the likelihood of the disease becoming confirmed and the supervention of dementia. . . . Epilepsy commencing in infancy and childhood is the least favourable for the arrest of the fits, and the most favourable for the production of the confirmed disease. The common type of epilepsy, or that commencing during puberty, is the most favourable form of epilepsy, both as regards the arrest of the seizures and the absence of mental infirmity. Adult epilepsy is unfavourable, but senile epilepsy is tractable. . . . Speaking in general terms, the earlier a case is brought under systematic treatment, the more hopeful the prognosis, and the greater the probability of improvement. The longer the interval between the attacks, the greater the prospect of arrest or improvement. The more frequent the attacks, the more common and profound the associated dementia. . . . The greatest percentage of arrests was in cases of the *grand mal*. Then followed the cases of the combined *grand mal* and *petit mal*, while the least favourable were the cases of the *petit mal* occurring alone. Freedom from mental impairment was found in both types, but to a minimal extent in those cases characterised by the *petit mal*, whether alone or in conjunction with the *grand mal*." Remissions extending over considerable periods of time are not uncommon in epilepsy. In this series of cases there were seven in which a remission of from two to five years occurred, five in which the patient was free from attacks for from five to eight years, and one case in which relapse occurred after an even longer period of respite, viz., fifteen years.

The author calls attention to two points in this connection: firstly, that long remissions occur under bromide administration, to be followed by a relapse when the drug is omitted; and, secondly, that a long remission may be broken by an accidental circumstance, such as a blow upon the head, a fall, child-birth, or an acute inflammatory disorder.

Regarding the curability of epilepsy, the author is of opinion that it is unsafe to regard any case as cured in which the seizures have not been in abeyance for at least nine years after the disease has become satisfactorily established. Conforming with this definition, 147 cases which have been under observation for at least nine years are available for conclusions as to the curability of the disease. Of these, 15 were arrested for nine years or more (10·2 per cent. of cases), and in 50 per cent. of the cases in which the disease was arrested, the arrest took place within one year.

Referring to the statistics of cure in the pre-bromide days, Dr Turner remarks that "the almost universal administration of the



bromides since their introduction in 1857, in the treatment of epilepsy, has in no way affected the variability of the results," and quotes figures in support of his statement.<sup>1</sup>

We presume that in this sentence Dr Turner means to expose the fallacy of relying on a comparison of these statistics, for he goes on to say that the exclusion of organic epilepsy by the later authors, and a discrepancy as to the definition of "cure," are to be taken into consideration in comparing the figures referred to.

At the end of his paper the author remarks on "the striking harmony between the results obtained and the percentage of 'cures' among those authors who have based their observations upon a clear definition of the term cure of epilepsy," and gives the following table:—

Author.	Definition of Cure.	Percentage.
Russell Reynolds .	Freedom for four to eight years.	10
Habermass . . .	Five to ten years.	10·3
Turner . . . .	Nine years.	10·2

It appears to us that a comparison of percentage of "cures" in the above table is misleading on account of the differences in the definition of "cure," for had Dr Turner defined "cure" as freedom for four to eight, or even five to ten years, his percentage of "cures" would undoubtedly have been very considerably higher than that of the two observers to whose results he refers.

EDWIN BRAMWELL.

<sup>1</sup> *Older statistics as to curability of epilepsy.*

Hufeland	gave 5 per cent. of cures.		
Russell Reynolds	„ 10	„	„
Trousseau	„ 13	„	„
Herpin	„ 50	„	„

*More recent statistics.*

Nothnagel	gives 4 to 5 per cent. of cures.		
Spratling	„ 5	„	„
Lähr	„ 6	„	„
Ackermann	„ 7·6	„	„
Dana	„ 5 to 10	„	„
Wildermuth	„ 8·5	„	„
Habermass	„ 10·3	„	„
Alt	„ 12·5	„	„

**ON THE OCCURRENCE OF MYASTHENIA WITH EXOPHTHAL-**

(55) **MIC GOITRE.** (Ueber das combinirte Vorkommen von Myasthenie und Basedow'sche Krankheit.) By RICHARD MEYERSTEIN, *Neurol. Centralbl.*, Dec. 1, 1904, p. 1089.

THE writer first gives a clinical account of the case.

The patient was an unmarried woman, æt. 33, who up till the time of this illness had good health. At Christmas of last year she noticed double vision which soon disappeared, but in April she complained of weakness in the neck, so that the head often fell forwards. The symptom also improved, but a few weeks later weakness in the arms appeared and she was unable to continue her work. She rapidly became fatigued, but her strength was restored by rest. Ptosis was also noticed. She suffered from palpitation, attacks of sweating and blushing.

On examination her condition was as follows:—

Considerable ptosis, complete paralysis of left external eye muscles, partial paralysis of the right eye, pupils normal. Exophthalmos of both eyes. The ptosis became rapidly increased after several attempts to open the eye. Weakness in the frontales and in the act of closing the eyes. Nothing abnormal about the lower face. No difficulty in speaking or swallowing. Thyroid considerably enlarged. In the upper extremities there was slight tremor in the hands, especially noticeable if the patient was tired. Weakness in the grasp, and if held out straight the arms gradually fell. The patient was unable to raise herself from the horizontal position. There was no appearance of fatigue in walking, but when the patient repeatedly extended the leg the amplitude of this movement was gradually less. No atrophy, no fibrillary tremors, and no ataxia or disorder of sensation. The knee-jerks were normal and not exhausted by being repeatedly produced.

Typical myasthenic reaction in the biceps and deltoid.

There was marked cardiac pulsation, apex and basal systolic murmurs and a pulse of 120.

Twenty-four hours before death the patient was suddenly seized with severe dyspnoea and great difficulty in speaking and swallowing. This attack proved fatal, respiration ceasing two minutes before the heart's action.

There was therefore a combination of myasthenia with certain symptoms of Basedow's disease, exophthalmos, enlarged thyroid, tachycardia, secretory and vasomotor disorders. Other symptoms such as typical tremor, diarrhoea, emaciation and diminished electrical resistance of the skin were absent.

The writer then refers to further cases in which this combination has been observed: Oppenheim's case with slight enlargement of the thyroid, tachycardia, tremor and diarrhoea; Kalischer's, with thyroid enlargement and exophthalmos, and others.

It follows that the combination of the symptoms of the two diseases is not rare. Is there in Basedow's disease a tendency for myasthenia to develop, or are the two sets of symptoms the result of the same cause? The writer inclines to the latter view, and calls attention to the fact that hypertrophy of the thymus has been found in both diseases.

W. B. WARRINGTON.

**KERNIG'S SIGN.** SAINTON and VOISIN, *Gaz. des. Hôp.*, août 27, (56) 1904, p. 949.

IN this article, the authors review the work which has been done on Kernig's sign since the first publication in 1883. Formerly thought to be pathognomonic of meningitis, it has since been shown to be present in such diverse conditions as sciatica, cerebral hæmorrhage, cerebellar hæmorrhage, cerebral abscess, tabes, hysteria, and certain toxic conditions which clinically simulate meningitis (meningism). The opinion that the presence of Kernig's sign in meningitis indicated that the disease was not tubercular has repeatedly been shown to be erroneous.

It was confidently stated at one time that the presence of this sign in toxic conditions always indicated that meningitis had set in, but it has since been repeatedly shown to be present in such conditions as pneumonia, uræmia, typhoid and other fevers, where lumbar puncture or post-mortem examination showed that no more than a simple congestion was present.

The authors discuss the nature and pathogenesis of the sign. It seems to be generally agreed that the essential part of the condition is a contracture of the flexor muscles of the limb. "The conception which seems to us to be the most simple consists in considering the sign of Kernig as a reflex phenomenon (with an elective localisation), a result of the bringing into play under various influences of the so-called excito-reflex cells of the cord. Thus it would be easy to explain its origin in the excitations of peripheral origin, like sciatica and lumbago; thus in hemiplegia it would give rise to a modification of the same kind; in cases of meningitis, it would depend either upon the local meningeal irritation, or upon a general modification of the cellular system of the posterior horns, under the influence of exogenous intoxication, and in certain particular cases of an intoxication of endogenous origin, without a meningeal reaction being indispensable."

In reading through this excellent résumé, one looks in vain for the evidences of diagnostic or other great value of the sign of Kernig, which we are led to anticipate in the opening words of the article:—"Since the discovery of lumbar puncture, and of the sign of Kernig, the clinical history of cerebro-spinal meningitis is become singularly more clear than before. . . . The sign of

Kernig has therefore taken a place of practical importance in daily practice. . . ." The resumé itself seems to refute these words, showing that the sign, though interesting as a clinical phenomenon, appears in such varied conditions which clinically simulate meningitis, that it has no real diagnostic importance.

STANLEY BARNES.

**WRITING AS THE CAUSE OF THE UNILATERAL POSITION OF**

(57) **THE SPEECH CENTRES.** (Das Schreiben als Ursache der einseitigen Lage des Sprachzentrums.) ERNST WEBER, *Zentralb. f. Physiologie*, Bd. xviii., No. 12, Sept. 10, 1904, p. 341.

ANATOMICALLY and developmentally no difference, as far as present methods of investigation indicate, can be traced between corresponding areas in the two cerebral hemispheres. Yet under normal conditions the function of speech is represented almost universally in the left hemisphere; it is necessary to destroy only a limited portion of the left cerebral cortex to produce a complete aphasia.

The received explanation of this curious fact is the general right-handedness of the genus Homo. The left hemisphere is more frequently and more profoundly called on to functionate because of the more frequent use of the right arm: and this is supposed to lead somehow or other to increased development of other, *inter alia*, the speech, centres on that side of the brain. Nevertheless, as a general rule, the difference between the daily work of the two arms is infinitely less than the corresponding difference in function between the right and the left speech areas.

For the author, then, the mere fact of the individual's right-handedness is insufficient to account for the placing of the speech centres in the left hemisphere. Right-handedness does not always reveal itself at an early period in the child's life: the observation is an old one that many children, up to a certain point, use either hand indifferently. The final determination of cerebral localisation of speech and allied centres depends on the fact that the child is *taught to write* with the right hand.

Aphasia in children, a not uncommon sequela, *e.g.* of typhoid, is usually of very short duration (two or three weeks). The common explanation is that the young growing tissues recover more easily than those of an adult. It is more probable that both speech centres are still capable of functioning, and that the one takes on the work when the other is destroyed. Various clinical cases have been recorded where aphasia occurred from a lesion in the right hemisphere, though the child was right-handed, or where the right centres maintained the function while the left were permanently damaged. Similarly in the case of illiterates, one has reason to believe that both sides of the brain are equally involved

in the process. The author quotes the instance of two right-handed illiterate Russians, who after a lesion in the right hemisphere (involving of course the left half of the body), entirely lost their ability to read, though they could still write. As probably they could read and write only to a very limited extent, the presumption is strong that the representation was bilateral, though not much developed, so that a lesion involving the right side of the cerebrum destroyed the function, and the left side being feebly developed was unable to undertake the work. That their writing, such as it was, was not affected, is explained by its unique representation in the left side of the brain. The researches of Mingazzini and Probst, among others, go to show that the musical faculty is bilaterally and equally represented, and is therefore independent of the right or left-handedness of the individual.

All these facts and analogies support the hypothesis that right-handedness *per se* is not sufficient to account for the exclusive localisation of the speech centres in the left cortex.

But the movements in writing are extraordinarily fine and sensitive, they vary vastly in different individuals; their cortical representation must be therefore all the more intricate and complete. And this writing centre, situated as it is on the left side, must have a distinct influence on the left speech centres, leading to their stimulation and development beyond those of the right. On reading, the visual word centre is excited and the stimulus passes *via* the hearing word centre to the motor speech centre; and the process is analogous for writing. This concentration, from writing and from reading, gives the left speech centre this opportunity for strengthening itself, so to speak, at the expense of its fellow on the right side.

In conclusion, the writer makes the interesting and suggestive remark, that the epoch-making discovery of Broca in 1862 became possible, although right-handed men had existed for thousands of years, only because the dissemination of the faculty of writing among all classes finally determined the localisation of the speech centres in the left cerebral cortex.

S. A. K. WILSON.

**STUDIES OF MOTOR APRAXIA. (Studien über motorische Apraxie.)**

(58) Prof. A. PICK, Prague. Pub. by Fr. Deuticke, Leipzig, 1905.

SINCE Hughlings Jackson, about thirty years ago, directed attention to the condition named by him imperception (asymbolia, agnosia), which he recognised in a case of cerebral tumour, much has been done in analysing this and kindred states. Like aphasia it is found to have a sensory and a motor form, and it is to illustrate the latter that Professor Pick has published the above memoir. The minute study of aphasia has yielded much information regarding the cerebral processes concerned with speech, and as a result of

this advance in knowledge, attention has lately been turned to the sphere of voluntary movement in general, speech being only one of its highly specialised forms. But the field of observation being so much wider and the phenomena so much more complex, it is not a matter for surprise that as yet no great advance has been made in mapping out this region. In the selection of cases whose investigation is here recorded, Professor Pick has limited himself to those which illustrate chiefly the motor form of apraxia, though it must be said that at times the symptoms showed evidences of sensory disturbance also. Hence the conclusions drawn are not entirely free from some elements of doubt.

The first case described is one in which the symptoms of motor apraxia were exhibited in an epileptic, and they are regarded as in part a manifestation of post-epileptic disturbance of consciousness. The patient was a man of 54, who for some years previously had suffered from convulsive seizures, chiefly at night. He had also been a great drinker for some time. After a succession of three severe fits in one night, he became quite insane, and was sent to the asylum. During the next month, at the end of which he died, he had no more fits. The method of examination consisted in placing before him certain common objects with which he was sure to be familiar, and observing what he did when asked to make use of them. The full particulars can, of course, only be obtained by reference to the original account, and all that can be said here is that, though the patient appeared to recognise the object and its use, he was unable to perform the necessary movements for making use of it. He had the idea of the acts, but he was unable to evoke the corresponding kinetic images. A frequently recurring phenomenon apparent in this and other cases was the persistent repetition of the same action in connection with quite different objects (perseveration). The attention could not, apparently, be readily turned from one set of actions to another. Nearly every day experiments were made to test his actions, and almost invariably some evidence of apraxia was found. A month after admission the patient died, but nothing abnormal was found in the brain, except sclerosis of the cornu ammonis.

The full details of other four cases are given, in all of which evidences of motor apraxia were apparent. In only one case was it possible to examine the brain, and in this there was found to be a glioma of the right second temporal convolution, with similar growths in the other hemisphere and in the pons. These morbid conditions were, unfortunately, of little use for purposes of localisation. Liepmann is inclined to the view that apraxia is due to some lesion in the parietal lobe, but it must still be considered uncertain whether the affection lies in the motor or the ideo-motor centres.

JAS. MIDDLEMASS.

**ON SOME RARE CONDITIONS IN GENERAL PARALYSIS:**

- (59) **APRAXIA, TRANSCORTICAL SENSORY APHASIA, SUBCORTICAL SENSORY APHASIA, SENSORY-MOTOR ASYMBOLY.** (Ueber einige seltene Zustandsbilder bei progressiver Paralyse, etc.) K. ABRAHAM (Dalldorf), *Allg. Zeitschr. f. Psych.*, Bd. 61, Heft 4.

ABRAHAM reports the cases of four paralytics who presented the complexes named above.

The careful investigation of focal symptoms in cases of general paralysis is often neglected because they are complicated by the general process, are often transitory, and are not expected to be easily correlated with the pathological findings. Where the patient after an apoplectiform attack recovers consciousness soon, the examination should be prompt, as the symptoms are likely to be transitory; where the loss of consciousness is more prolonged, the focal symptoms will also be of longer duration. The first case given by the author was a general paralytic, aged 34, who was able to be employed in the shoemaking department of the hospital without making serious mistakes. On the 14th October he had an apoplectiform attack, quickly recovered consciousness; the right forearm was found to be weak; patient did not speak nor obey commands. On the 15th October the paresis of the forearm had almost completely disappeared. Patient now named objects correctly; understood what was said to him; his only speech-defect was his articulatory defect. He executed correctly orders which involved the whole body; but when asked to raise his hands, clench his fist, open a door, he made quite inappropriate gestures and movements, showing, however, no ataxia. Similarly he failed to imitate simple movements involving the upper extremities. The patient's receptive apparatus was intact; he understood what was said to him, recognised objects, had no considerable defect in the sense of touch, no ataxia. The disorder therefore was in the motor sphere, and consisted in an apraxia or motor asymboly. On the 25th October there was no trace of this symptom left.

The second case was a paralytic aged 38, who, after a slight apoplectiform attack, was for two days unable to understand what was said to him. He recognised objects, as was seen from his conduct; he never made inappropriate movements; he imitated promptly gestures and movements. He was able to repeat fairly well words said to him, but was unable to understand spoken or written commands. His speech was extremely paraphasic. The complex was that of Wernicke's transcortical sensory aphasia.

The third case presented a similar interference with the receptive apparatus, while the motor apparatus remained normal. Spontaneous speech was paraphasic, and patient could not repeat

words spoken ; but his general reactions to environment were normal. He was unable to understand spoken and written orders, and was essentially an example of Wernicke's subcortical sensory aphasia.

The fourth case was that of a paralytic aged 31, who on admission was almost quite unintelligible and could only carry out a few orders, and who finally developed a complete aphasia ; at first he was able to pick out objects named and imitate some movements, but he later showed marked inability to understand speech, and finally gave absolutely no reaction to orders of any kind. On admission he had shown partial apraxia, making most inappropriate movements of various kinds ; latterly appropriate movements were rare, and finally absent. There were therefore present both a sensory and a motor asymboly.

C. MACFIE CAMPBELL.

**MYOTONIA WITH MUSCULAR ATROPHY.** (*Myotonie avec (60) Atrophie musculaire.*) LANNOIS, *Nouv. Icon. de la Salpêtr.*, Nov.-Dec. 1904, p. 450.

THE case which is described might equally well have been called one of progressive myopathy with hypertonus, or Thomsen's disease *fruste* with muscular atrophy.

A man, thirty-three years of age, with no hereditary nervous affection, neither syphilitic nor alcoholic, begins to suffer from a "steppage" gait due to atrophy of the leg muscles, from lightning pains and absence of knee-jerk, which lead to a diagnosis of tabes. But simultaneously appear myotonic phenomena absolutely analogous to those of Thomsen's disease, though more limited. Slow aggravation of symptoms, and progressive muscular asthenia with the myotonic electrical reaction, characterise the condition at present.

Apparently one has to do with a superposition of myopathic and myotonic symptoms. At least a dozen similar observations exist, pointing to the differentiation of a clinical and perhaps anatomo-pathological entity, viz., atrophic myotonia. Möbius has recently maintained that there is a close connection between Thomsen's disease and pseudo-hypertrophic paralysis, while Oddo similarly has attempted to establish a relation between myopathy, myotonia, and myoplegia ("paroxysmal" or "periodic" family paralysis).

S. A. K. WILSON.

**VASOMOTOR SYMPTOMS IN AN HYSTERICAL SUBJECT.** (61) (*Troubles vasomoteurs chez une hystérique.*) GÉNÉVRIER *Nouv. Icon. de la Salpêtr.*, Nov.-Dec. 1904, p. 459.

A CASE of *grande hystérie*, in which, among other more or less typical manifestations, appeared from time to time cedema,



cyanosis, and local syncope of the extremities, local exudations of sweat, and large plaques or blotches of irregular distribution over back, abdomen, legs, hands, livid at first and slightly raised, changing in the course of two or three days to bullous areas, ending in cicatricial "pseudo-keloids." The condition is probably, in its protean manifestations, an intense vasomotor neurosis, leading to definite organic alterations. S. A. K. WILSON.

### PSYCHIATRY.

#### THE DEFINITION OF CERTAIN SYMPTOMS OF DEMENTIA

(62) **PRÆCOX.** (*Zur Auffassung gewisser Symptome der Dementia præcox.*) E. STRANSKY, *Neurol. Centralbl.*, 1904, pp. 1074 and 1137.

THE first part of this paper is concerned with the author's claim to the priority of recognising certain symptoms which he considers characteristic of dementia præcox. These symptoms consist in a loss of the unity of the processes of cognition, feeling, and volition, which normally characterise the healthy mind. It is not peculiar to dementia præcox, but it is most frequently and characteristically met with in that disease. The necessity for this claim to priority arises out of the fact that in a recently published article Weber attributes it to Gross. The origin of the mistake appears to have been a difficulty in understanding the exact meaning of certain terms used by the two writers in a slightly different sense. Until the contentions of each have been proved of value, and that time has not yet arrived, it seems unnecessary to spend so much time in upholding one claim against the other.

The second part of the paper gives an account of an interesting case of what the author considers to be dementia præcox. He records certain symptoms which may be explained on the ground of what is called intrapsychical inco-ordination or ataxia. He distinguishes this from Wernicke's mental dissociation on several grounds. Both theories, however, are purely speculative, and their truth and value have yet to be proved.

JAS. MIDDLEMASS.

#### ON THE NAME "DEMENTIA SEJUNCTIVA." (*Zur Nomenclatur*

(63) "*Dementia sejunctiva.*") O. GROSS, *Neurol. Centralbl.*, 1904, p. 1144.

THIS is Gross's reply to Stransky's paper, noticed above. He has suggested the substitution of this term for dementia præcox. At the outset he notes that dementia has gradually been altering its meaning from a state to a process, and it is in the latter sense that he uses it. He argues that, where possible, the name of a disease

should give some idea of its etiology ; but when this is not possible, that it should indicate its most prominent symptom. It is admitted that mental dissociation is present in many forms of mental disease, but that this symptom, in cases of dementia, is most pronounced in those which have previously been known under the name of dementia præcox. His contention is not that Stransky's theory of mental inco-ordination is the same as Wernicke's, but necessarily is included in the latter as the greater includes the less. He regards his own theories as only an expansion of those of Wernicke.

JAS. MIDDLEMASS.

**ON STATIONARY GENERAL PARALYSIS.** (*Zur Frage der (64) stationären Paralyse.*) C. WICKEL, *Centralbl. f. Nervenh. u. Psych.*, Sept. 1904.

By stationary paralysis Wickel does not mean a remission of the disease with amelioration of the symptoms, but the arrest of the morbid process at a more or less advanced stage of the disease, the patient presenting the same clinical picture for a prolonged period. He gives the observations of three cases of advanced general paralysis, who for a period of  $8\frac{1}{2}$ ,  $7\frac{1}{2}$ ,  $5\frac{1}{2}$  years respectively have presented the same condition. The diagnosis was clear; post-traumatic dementia, alcoholic dementia, and diffuse brain-syphilis could be excluded. The author considers that several cases of so-called "stationary paralysis" probably are cases of diffuse brain-syphilis.

C. MACFIE CAMPBELL.

**ON GANSER'S SYMPTOM.** (*Ueber das Gansersche Symptom.*) (65) R. HENNEBERG, *Allg. Zeitschr. f. Psychiat.*, Bd. 61, Heft 5.

HENNEBERG gives the history of thirteen cases which showed this symptom, consisting in the patient giving to a simple question a false answer, which, however, is closely related to the true answer: thus the patient will call a postage-stamp "paper," a match-box a "pill-box," a candle "wick." This symptom occurs in hysterical psychoses of the most varied nature, but is always a chance element in the picture. Typical cases of catatonia may present the symptom, which Nissl considers an expression of negativism: Ganser considers that in such cases hysteria is a complication of the catatonia. The author considers that it may occur in cases of catatonia with no hysterical traits. Normal individuals under certain conditions, such as sleepiness, embarrassment, petulance, present a similar phenomenon, which is also one of the most natural resorts of those who wish to simulate insanity. Ganser's symptom is five times more common in criminal cases than in other cases: even in hysteria the desire to appear ill probably favours its production. The patients who give these inappropriate answers to very simple

questions never make spontaneously such absurd statements. The symptom occurs in so many conditions that it has no special diagnostic value as yet; one cannot be too suspicious of it in criminal cases, as the more eagerly one tests for it, the oftener it is forthcoming.

C. MACFIE CAMPBELL.

**TWO CASES OF GENERAL PARALYSIS WITH BRAIN-  
(66) SYPHILIS : PSEUDO - PARALYSIS SYPHILITICA OF  
JOLLY.** (Über zwei Fälle von Dementia paralytica mit  
Hirnsyphilis.) With 9 photographs. RENTSCH, *Arch. f.  
Psych. u. Nervenh.*, 1904, Bd. 39, Heft 1.

JOLLY grouped under the term pseudo-paralysis syphilitica cases which clinically could not be differentiated from general paralysis, but which on post-mortem examination presented, in addition to the diffuse degenerative changes of general paralysis, local changes in the brain of undoubtedly syphilitic origin. Rentsch publishes the clinical observations and post-mortem reports of two cases which came under Jolly's group.

In both cases the characteristic cortical changes of general paralysis were found; in addition, the first case showed a gummatous arteritis limited to some of the basal cerebral arteries, but sparing the internal carotids, while the second case presented a small gumma at the base of the brain.

The author emphasises the importance of such cases in establishing the etiological relationship of syphilis to general paralysis.

C. MACFIE CAMPBELL.

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## Review

**TEXT-BOOK OF MENTAL DISEASES.** (Trattato delle  
malattie mentali.) EUGENIO TANZI. Società Editrice  
Libreria, Milano, 1905. Pp. 764. 20 lire.

THE Psychiatric Clinic of Florence already enjoys a world-wide reputation. For many years, and at least ever since its organ the *Rivista di Patologia nervosa e mentale* began to appear in 1896, it has held a foremost place as a centre of neurological research. To the credit of this school there stand not only the many notable original papers of its leader, but also the splendid series of experimental, anatomical, and other researches, through which Lugaro has done so much to advance neurological and mental science, as well as numerous important contributions by Levi, Catòla, Camia, Rebizzi and others. The publication of this book, which must naturally reflect the achievements of the school from which it

comes, is therefore no ordinary event in the progress of psychiatry. Those who have been led to expect much of the work will not be disappointed. Beyond question Professor Tanzi has done more than simply add to the number of existing treatises upon insanity; he has written what is certain to rank in medical literature as a great book. He has given to the scientific world a magnificent synthesis of the facts and hypotheses that constitute modern psychiatry, as they are viewed by one who is not only an acute observer and original thinker, but who has at the same time the gift of clear and forcible exposition. The lucidity of the writing, even when the most difficult subjects are being dealt with, is indeed one of the striking features of the book. There are doubtless many to whom the present superabundance of psychiatric literature, and the interminable conflict of opinion which it shows, have brought bewilderment rather than enlightenment, and left the impression that the pathogenesis of insanity is still little more than a matter of vague speculation. Such persons will find it refreshing to turn to the clear and convincing exposition of mature opinions contained in this book. Its pages contain many striking and surprising generalisations before some of which the reader will be apt to pause to ask, Has psychiatry really advanced so far? The evidence adduced will rarely admit of a negative answer, and a careful perusal of the book can hardly fail to bring conviction that modern psychiatry is not the backward science it is often alleged to be, but that, with the ascertained facts marshalled and interpreted as they are in this book, it is one of the most advanced and richest departments of medical knowledge.

A brief outline of the contents of this most important work must suffice here. Chapter i. deals with the seat of the psychical processes. The data of physiology, experimental anatomy, embryology, human pathology, and normal anatomy are successively reviewed, and finally the positive evidence in favour of the existence of psychical centres is discussed. In the next chapter, the causes of mental diseases are minutely analysed. The large rôle assigned to external influences acting through the production of disorders of general metabolism will probably strike many as remarkable. The author states the conclusions that among the causes of insanity the external have greater influence than the internal, and that among the external causes somatic disorders and social *disarmonie* are more to be feared than intellectual overwork, the action of which is of no account except when accompanied by hygienic errors, or by depressing emotions, which entail trophic disturbances of the whole organism. The next chapter deals with the anatomical substratum of mental diseases, a subject towards the elucidation of which the original researches carried out in the Laboratory of the Psychiatric Clinic of Florence have contributed

so very largely. The immediately succeeding five chapters treat of the psychological problems concerned with insanity. Sensibility, ideation, the memory, the sentiments and the movements, and other external reactions, are in turn considered. It is in dealing with these difficult subjects that the author's independence of judgment and remarkable power of lucid exposition are perhaps most conspicuously shown. Chapter ix. is upon the classification of mental diseases. Previous classifications, more particularly those of Krafft-Ebing, Morselli and Kraepelin, are examined at considerable length, and the principles of a new classification, based essentially upon etiology, are then very fully expounded. This classification of Professor Tanzi's seems so important that it may be useful to give it in full here.

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|--|---|---|---|
| POISONINGS.                                    | { | 1. Pellagra.  |   |
|  |   | 2. Alcoholism.  |   |
|  |   | 3. Morphinism, cocaineism.                                |   |
|  | { | 4. Amentia.   | { Hallucinations.                       |
|  |   |   | { Apathetic.                            |
|  |   |   | { Slight (acute systematised insanity). |
|  |   |   | { Very severe (acute delirium).         |
| TOXIC INFECTIONS<br>AND<br>AUTO-INTOXICATIONS. | { | 5. Uremic psychoses.                                      |   |
|  |   | 6. Thyroid psychoses.                                     | { Acquired myx-<br>œdema.               |
|  |   |   | { Cretinism (endemic<br>and sporadic).  |
|  |   |   | { Basedowism.                           |
|  |   | 7. Progressive paralysis.                                 |   |
|  | { | 8. Infantile cerebropathy (acquired idiocy).              | { Cerebral tum-<br>ours.                |
|  |   |   | { Head trauma-<br>tism.                 |
| ENCEPHALOPATHIES.                              | { | 9. Cerebropathies of adults.                              | { Cerebral<br>syphilis.                 |
|  |   |   | { Apoplectic<br>dementia.               |
|  |   |   | { Senile demen-<br>tia.                 |
|  | { | 10. Melancholia.  |   |
| AFFECTIVE PSYCHOSES.                           |   | 11. Mania.  |   |
|  |   | 12. Circular psychoses.                                   |   |
| CONSTITUTIONAL<br>NEURO-PSYCHOSES.             | { | 13. Constitutional neurasthenia (obsessive<br>psychoses). |   |
|  |   | 14. Hysteria.   |   |
|  |   | 15. Epilepsy.   |   |

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|-----------------------------------|---|--|
| DEMENTIA PRÆCOX.                  | { | 16. Hebephrenic form.                                |
|                                   | { | 17. Catatonic form.                                  |
|                                   | { | 18. Paranoid form.                                   |
| DEGENERATIVE<br>MENTAL ANOMALIES. | { | 19. Perversion of the sexual instinct.               |
|                                   | { | 20. Constitutional immorality.                       |
|                                   | { | 21. Paranoia.  |
|                                   | { | 22. Intellectual feebleness (hereditary imbecility). |

Chapters x. to xxv. deal with the individual forms of mental disease in the order of the above classification. There is here presented to the reader a series of masterly studies, in which symptomatology, pathology and treatment are in each instance very fully and clearly described. The book ends with a chapter upon Asylums, which includes an eloquent appeal for reform, of which it may be said other countries stand quite as much in need as Italy.

The publication of two such splendid treatises as those of Professor Bianchi and Professor Tanzi, which have been completed almost simultaneously, certainly reflects the highest credit upon Italian psychiatry. Both are books destined to exert an influence far beyond the country in which they have had origin, and it is therefore satisfactory that they are to be made more accessible to British alienists and students by means of translations.

W. FORD ROBERTSON.

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# Review of Neurology and Psychiatry

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## Original Articles

### THE PROGNOSIS OF DISSEMINATED SCLEROSIS.

By BYROM BRAMWELL, M.D., F.R.C.P.E.,

Physician to the Edinburgh Royal Infirmary, etc.

IN the great majority of cases, disseminated sclerosis, sooner or later, causes death; consequently the ultimate prognosis is extremely unfavourable.

The duration of the disease varies greatly in different cases. The course is usually slow and chronic, the disease may last for twenty or more years before the fatal termination is reached. In rare cases, the disease pursues a rapid course. It is probable, I think, that in a few rare and exceptional cases the disease is permanently arrested, and a cure, or what is practically speaking a cure, takes place.

In some cases, the symptoms, once they are developed, pursue a progressive course from bad to worse; but, in many cases, the downward progress is from time to time interrupted by periods of improvement or complete remission of the symptoms. In some cases, the symptoms (giddiness, numbness, diplopia, inco-ordination, loss of power, dimness of vision, nystagmus, volitional tremor, speech affection, etc.) entirely disappear or almost entirely disappear for a time. These periods of improvement and remission are very deceptive, for they may lead one to give a favourable prognosis, and, if any special plan of treatment has been adopted, to attribute the amelioration to that treatment. Unfortunately, experience shows that, in the great majority of cases of

this kind, the improvement is merely temporary. Everyone who has had much experience of disseminated sclerosis knows that, although the patients often improve, and in rare instances apparently for a time get quite well, it is only in very rare instances that the improvement is lasting. The great majority of cases sooner or later relapse. Nevertheless, I doubt whether one is justified in saying that the improvement is never permanent and that a cure never takes place. In rare cases the improvement is so marked (the symptoms completely disappearing) and the remission so prolonged, that one ventures to hope that in such cases a permanent arrest or cure has taken place.

In a recent clinical lecture on "Remissions and Relapses in Disseminated Sclerosis" (*Lancet*, July 16, 1904, p. 131), Dr Buzzard has recorded some cases in which an unusually long remission of all the symptoms occurred.

In a disease like disseminated sclerosis, in which the symptoms may entirely disappear for a time (for several months or years), it seems reasonable to suppose that permanent arrest and a cure would occasionally occur. The wonder is that permanent arrest and cure do not more frequently take place. Unfortunately, experience shows that permanent arrest is quite exceptional; indeed most authorities seem to doubt whether a cure ever takes place.

In some of the rare cases in which the symptoms completely disappear and a (? permanent) arrest of the disease seems to take place, optic atrophy or the Babinski sign remains; but these conditions are to be regarded as results rather than active manifestations of the disease.

Many authorities believe that the patches of sclerosis, which are the pathological substratum of disseminated sclerosis, are due to the irritation produced by some form of toxin, carried to, and distributed through, the nervous tissues by the blood-vessels. The recurrence from time to time of the symptoms after periods of improvement and remission is very suggestive of repeated intoxications. If disseminated sclerosis is due to a toxin, the toxin, whatever it is, is probably produced within the body. It seems much more difficult to suppose that fresh doses of the toxin are introduced again and again into the body from without during a long period of years.

Now, if the exacerbations and relapses in disseminated

sclerosis are due to recurring intoxications, it is not unreasonable to suppose that, in some cases, the development of the toxin from within or the absorption of the toxin from without may cease, and that a permanent arrest and cure may occur.

Dr Buzzard has suggested that the poison is perhaps analogous to the syphilitic poison, which, once it has been introduced into the body, is apt to produce recurring lesions during a long period of years.

RESULTS OF TREATMENT IN 110 CASES OF DISSEMINATED SCLEROSIS.—So far as I know, there are no detailed and definite statistics showing the duration and results of treatment in any considerable number of cases of disseminated sclerosis. I have consequently been at great pains to follow up all of my cases, and to ascertain the duration of the disease and the present condition of the patients. In the 110 cases which I have recently analysed, the results up to the present date (January 1905) are as follows:—35 of the 110 cases have died; 61 cases are known to be still alive; and in 14 cases the result is not known. Of the 61 cases which are known to be still alive, 33 are markedly worse, 16 are *in statu quo*, 8 are more or less improved, and 4 are quite well. (See Table I.)

Table I. Showing the Results in 110 Cases of Disseminated Sclerosis.

Died . . . . .	35 cases
Still alive—	
Worse . . . . .	33
<i>In statu quo</i> . . . . .	16
More or less improved . . . . .	8
Quite well . . . . .	4
Not known . . . . .	14 „
	<hr/>
	110 „

THE TOTAL DURATION OF THE DISEASE SINCE THE COMMENCEMENT UP TO THE PRESENT DATE (JANUARY 1905).—This was definitely ascertained in 96 cases: the average duration of the disease in these 96 (fatal and non-fatal) cases is, up to the present date, 10 years and 5 months. In one case the patient, who is still alive, has had the disease for 33 years, and in no



less than seven cases the patient has been affected with the disease for more than 20 years. (See Table II.)

*Table II. Showing the Total Duration in 96 Cases of Disseminated Sclerosis in which the result was known in January 1905.*

$1\frac{1}{2}$ year	1 case	14 years	4 cases
2 years	3 cases	15 „	5 „
3 „	5 „	16 „	2 „
4 „	3 „	17 „	2 „
5 „	6 „	18 „	2 „
6 „	11 „	19 „	3 „
7 „	7 „	20 „	1 case
8 „	8 „	21 „	1 „
9 „	4 „	23 „	1 „
10 „	9 „	24 „	1 „
11 „	6 „	26 „	1 „
12 „	4 „	29 „	1 „
13 „	4 „	33 „	1 „
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Total cases			96

THE TOTAL DURATION IN 35 FATAL CASES OF DISSEMINATED SCLEROSIS.—In the 35 cases in which the disease proved fatal, the average duration was 7 years and 9 months; the shortest duration was 7 months, and the longest duration was 21 years. The exact duration in each of the 35 fatal cases is shown in the following table:—

*Table III. Showing the Exact Duration in 35 Fatal Cases of Disseminated Sclerosis.*

Under 1 year	1 case	} 10 cases
2 years	3 cases	
3 „	4 „	
4 „	2 „	
5 „	2 „	} 13 cases
6 „	5 „	
7 „	2 „	
8 „	3 „	
9 „	1 case	

Under 10	„	2 cases	} 8 cases
11	„	3 „	
12	„	2 „	
13	„	1 case	
15	„	2 cases	} 3 cases
18	„	1 case	
21	„	1 „	} 1 case

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35

From this table it will be seen that in 10, or 28·5 per cent. of the 35 fatal cases the disease terminated in less than 5 years; in 23, or 65·7 per cent. of the 35 fatal cases in less than 10 years; and in 31, or 88·5 per cent. of the fatal cases in less than 15 years.

The following are notes of two typical and two atypical cases of disseminated sclerosis in which recovery, or complete disappearance of the symptoms for such a length of time as to suggest apparent recovery, has taken place :—

**CASE 1. DISSEMINATED SCLEROSIS : RECOVERY.** Female, single, aged 23, was seen on 7th March 1898.

**HISTORY.**—The patient, who is a bright girl and not naturally nervous or hysterical, enjoyed robust health until sixteen months ago. For a year after this date she did not feel “up to the mark,” but there were no definite signs of disease. Four months ago, giddiness, ptosis, squint, double vision, and numbness in the left arm and both legs developed. After lasting for three weeks, these symptoms disappeared and after a time returned. She has got much worse of late.

**STATE (7th March 1898).**—The patient complains of giddiness. The gait is very unsteady—spastic and ataxic; the calves are soft and flabby; the movements of the upper extremities are inco-ordinate, but there is no volitional tremor. Nystagmus, great exaggeration of both knee-jerks, ankle-clonus (marked on the right, slight on the left side) are present. The plantar reflex is very active (the Babinski sign was not looked for); there is some difficulty in micturition; the bowels are markedly constipated. The pupils are equal and active to light and on convergence; vision is unaffected; the optic discs are quite normal—no atrophy, no neuritis; there is no speech affection (it developed later); no headache; no vomiting; no hysterical symptoms; the menstruation is normal.

**DIAGNOSIS.**—There was no evidence of a coarse intracranial lesion, such as a cerebellar tumour or meningitis—no headache, vomiting, optic neuritis. The diagnosis was disseminated sclerosis; the marked improvement and subsequent relapse seemed to confirm this view of the case.

**SUBSEQUENT PROGRESS OF THE CASE.**—In July 1898, the doctor wrote to say :—“I have to report great and continued improvement. On four occasions the patient has had attacks of headache and vomiting; these attacks were always associated with dyspepsia and a foul tongue, and were evidently gastric and not cerebral in character. The patient's walking is much better—

she has walked a distance of three or four miles without fatigue ; very slight unsteadiness in gait is still present ; the nystagmus is now hardly detectable ; the exaggeration of the deep reflexes has almost disappeared ; the patient's friends and relatives notice a certain forgetfulness and a tendency to be childish in her ways and thoughts."

*On 2nd September 1898*, I again saw the patient and made the following note :—She looks well and states that she feels perfectly well ; there is still some slight giddiness ; her memory is not so good as it used to be ; her mother occasionally notices some dragging of the leg ; the speech is thicker than it used to be ; her mother states that when the symptoms were at their worst, speech was markedly affected ; the knee-jerks are still exaggerated ; there is no ankle-clonus ; slight nystagmus is still present ; the patient is more emotional than she used to be.

*On 29th May 1899*, the doctor wrote :—"The symptoms have relapsed ; double vision ; internal strabismus of the right eye ; thickness of speech ; staggering gait ; great exaggeration of the knee-jerks and ankle-clonus ; a feeling of numbness all over the body ; drawing of the mouth to the left side ; want of taste on the right side of the tongue ; diminished tactile sensibility round the angle of the mouth on the right side. The symptoms vary very much from time to time."

*In June 1900*, the patient felt and seemed to be quite well ; she married without medical sanction.

*In June 1904*, the doctor wrote to say :—"The patient still keeps quite well ; she has had no children ; there has been no return of the symptoms since marriage ; the patient is stouter, and says she never was so strong in her life as she is to-day."

*In January 1905*.—The patient keeps quite well.

**NOTE.**—There can, I think, be no doubt as to the diagnosis in this case. The history (the mode of development and nature of the early symptoms—temporary and recurring giddiness, squint, diplopia, numbness, difficulty in walking), is very striking and characteristic. The symptoms, when I saw the patient, seemed to me to conclusively point to disseminated sclerosis, viz. : giddiness, ataxic-paraplegia, inco-ordination in the upper extremities, nystagmus, ankle-clonus, great exaggeration of the knee-jerks, some difficulty in micturition, and (subsequent) speech affection.

**CASE 2. DISSEMINATED SCLEROSIS : RECOVERY.**—Female, aged 49, married, was seen on 23rd June 1902, complaining of numbness and weakness in the left hand and arm.

**PREVIOUS HISTORY.**—In 1891, after influenza and rheumatism, the patient had numbness and loss of power in the left hand ; she has had many attacks of the same sort since. In 1892, numbness and loss of power developed in the feet and legs. In the spring of that year (1892), the nervous symptoms became much more marked, she was very ill, and was seen in consultation by Sir Christopher Nixon of Dublin, who gave a very bad prognosis.

*In October 1893*, she saw Mr George Berry on account of loss of vision, first in one eye, then in the other. There have been many ups and downs in the case since.

**STATE (23rd June 1902).**—On examination, the only objective signs indicative of disease which I was able to detect, were marked exaggeration of the knee-jerks, particularly the left, the Babinski sign on the left side, some (slight) inco-ordination of the left hand, and very slight inco-ordination of the right hand on performing the finger-nose-test. There was no nystagmus ; no volitional tremor ; no speech affection (the articulation had been affected) ; no difficulty with urination ; no more constipation than one might easily get in a woman (corrected by a dinner pill) ; no ankle-clonus ; and no defect of memory.

I wrote to the doctor saying that the history (repeated attacks of numbness and loss of power, and on two occasions of dimness of vision, *all recovered from*) was very suggestive of disseminated sclerosis, though it might of course be merely the result of functional disturbance; that the Babinski sign and the inco-ordination in the hands pointed strongly in the same direction; that I regarded the Babinski sign as evidence of organic disease, and that in conjunction with the history it pointed strongly to disseminated sclerosis; that Sir Christopher Nixon, who saw the patient when she was at her worst, in the year 1892, seemed to have taken a very grave view of the case; that I had written to him and also to Mr Berry, who saw the patient because of the dimness of vision in the year 1893, to try and find out what they thought of the case; that I suspected that the symptoms were due to disseminated sclerosis, and that I should look forward with interest to what Sir Christopher Nixon and Mr Berry said regarding the condition when they saw the patient ten years ago.

On 24th June 1902, Mr George Berry wrote:—"On 30th October 1893, the patient had V. =  $\frac{20}{100}$  left eye; no ophthalmoscopic changes; no pain on pressing the eye back into orbit. My diagnosis was *retrobulbar neuritis* (peripheral and probably rheumatic)."

On 28th June 1902, Sir Christopher Nixon wrote:—"My recollection of Mrs —'s case is that it was one of multiple cerebro-spinal sclerosis, and I am surprised to hear that she got, at any time, completely rid of her symptoms. She had, at the time I saw her, spastic gait, intention tremor, slight degree of nystagmus, and some attacks of giddiness. I saw her, I think, twice."

On 24th June 1904, the patient's doctor wrote me saying:—"She is quite well, and has been quite well since you saw her in June 1902; the knee-jerks are still markedly exaggerated; the Babinski sign was not obtained on June 23rd." And again, on 16th January 1905, "the patient remains quite well."

**CASE 3. ATYPICAL DISSEMINATED SCLEROSIS: APPARENT RECOVERY.**—Female, single, aged 26, was seen on 25th July 1899.

**HISTORY.**—The patient, who is a somewhat nervous girl, enjoyed good health until three and a half years ago (*January 1896*); she then gradually lost the sight of the left eye: she consulted Dr George Mackay, who found optic neuritis present in the left eye, with marked impairment of vision. Under treatment, the optic neuritis gradually disappeared and vision was to some extent regained. In August 1898, she lost vision in the right eye. On 30th August 1898, optic neuritis was found to be present in the right eye; under treatment this gradually improved. Her medical man (Dr Edward Carmichael) has kindly sent me the following report from Dr George Mackay on the condition of the optic discs and vision:—"Miss C. is hypermetropic and astigmatic (about the dioptré of each in each eye). In the beginning of January 1896, she began to have pain above the left eyebrow and in the temple.

"On 15th January 1896, she first came to me with left optic neuritis, very vivid hyperæmia of the disc substance, but not much tortuosity of veins nor much swelling of the disc. Some tenderness on backward pressure of the left globe.

"R.V. with glasses =  $\frac{3}{8}$  good field.

"L.V. with glasses = hand movements only in the outer part of the field.

"She was ordered eye rest, leeches to the temple and sod. salicyl. Later, the temple was blistered, and pot. iod. with hyd. perchlor. given.

"By 11th February, L.V. had returned to  $\frac{3}{8}$ .

"By 7th March, exudation gone, disc pale and partially atrophied, but good field for hand movements.

"She returned to work and did not consult me again until 29th October 1897, when I found vision unchanged. She is naturally high-coloured, and

her right disc always looked hyperæmic, but showed no sign of inflammation until 30th August 1898, when, in my absence, Dr Matthew (my assistant) found the right disc hazy, but R.V. still =  $\frac{3}{4}$  with difficulty.

"On 15th September I found R.V. with glasses =  $\frac{3}{4}$ . No pain nor tenderness. A decided scotoma for colours to the inner side of the point of fixation and across the middle line, and diagnosed a retrobulbar neuritis. The same kind of treatment was again adopted, but *sod. salicyl.* pushed more freely and perspiration encouraged by hot pack.

"By 31st January 1899, R.V. returned to  $\frac{3}{4}$  partly, and by 7th April continued the same, but trace of scotoma for colours still present. Right disc hyperæmic, but not obscured by exudation. L.V. had improved to  $\frac{1}{2}$  partly, and she seemed much better in all respects. Complained that her sight became worse on exertion. I have not seen her since, and much regret to get your report of this new development."

A month ago (in June 1899) she was accidentally knocked over in the street; since then she has complained of numbness in the forefinger and thumb of the right hand, difficulty in writing, numbness and weakness in the legs, and unsteadiness and difficulty in walking.

STATE.—On examination, marked optic atrophy was found to be present in the left eye; the right disc seemed normal; the knee-jerks were both markedly exaggerated; slight ankle-clonus and a double extensor response were present on both sides; marked Rombergism; inco-ordination in the right hand, and some volitional tremor in the right hand. The general health was good; there was no giddiness, no headache, no vomiting, no nystagmus, no speech affection, no urinary derangement, no objective disturbance of sensation, no loss of the muscular sense. The hands, feet and face were cold and blue, especially on exposure to cold; menstruation was regular and natural. There were no signs of congenital or acquired syphilis.

DIAGNOSIS.—I diagnosed the case as one of commencing disseminated sclerosis, and my son—Dr Edwin Bramwell—who saw the case with me, agreed with this diagnosis.

Arsenic and nitrate of silver were prescribed.

SUBSEQUENT PROGRESS OF THE CASE.—10th June 1902.—Much better. The only thing she now complains of is blueness and coldness of the face, hands and feet, and sometimes a numb feeling in the hands and feet. The knee-jerks are still exaggerated, ankle-clonus is still present, especially in the right foot, the plantar reflex shows marked extension in the right foot, no toe movement in the left; slight inco-ordination and some volitional tremor still present in the right hand. Distant vision in the right eye =  $\frac{1}{12}$ , in the left,  $\frac{3}{8}$ . Near vision, with glasses, right = 0.5, left = 0.5 (imperfect). The left disc is still very pale.

26th January 1905.—Patient says that she has been quite well since last seen. Face, hands and feet still blue and cold; knee-jerks still exaggerated; no ankle-clonus; extensor response present in the right, absent in the left foot; left disc still markedly paler than the right. Dr Carmichael informs me that the medicine which seemed to do her most good, and to which she herself attributes her recovery, was valerian.

CASE 4. ATYPICAL DISSEMINATED SCLEROSIS: APPARENT RECOVERY.—Female, single, aged 28, was seen on 30th June 1897. Not naturally nervous or hysterical.

HISTORY.—In October 1895, after long nursing of her mother, lost power in the legs and had difficulty in walking; power regained in a fortnight; remained well for six weeks, then suddenly became lame again. Has had double vision and dimness of vision more than once, and has noticed that objects jump up and down when she looks at them (nystagmus); has also had more than one attack of loss of power in the arms and numbness in the legs. Last winter she suffered from giddiness, loss of power on one side of the face

and difficulty in articulation. About a week ago she completely lost power in both legs and was unable to make water.

**STATE.**—General health good. Walks with a perceptible drag, especially of the left leg; knee-jerks markedly exaggerated; ankle-clonus present (the Babinski sign was not then known). No volitional tremor, no speech affection (at present), no optic atrophy.

**SUBSEQUENT PROGRESS OF THE CASE.**—*14th July 1902.* Improved after last visit, and was so well that she could dance. A year later the loss of power in the legs returned without obvious cause.

**PRESENT CONDITION.**—Feels in perfect health, occasional giddiness; memory not so good as it used to be, temper more irritable and excitable, more emotional; urination affected, often precipitant, occasional dribbling; some numbness in the legs, occasionally numbness in the hands; complains of coldness of the feet; knee-jerks markedly exaggerated; no ankle-clonus; plantar reflex—right flexion, left extension; abdominal reflexes both absent. No nystagmus; no speech affection; no optic atrophy.

*4th July 1904.*—In November 1902, after a strain nursing her father, felt giddy; memory became more impaired; walking again became affected. Says she is now quite well again. Right knee-jerk exaggerated, left normal; plantar reflex—double extensor response from ball of toes; flexion from the sole; urination not now affected; optic discs normal; no other symptoms.

*24th January 1905.*—Writes to say she is quite well.

The following are the notes of a typical case of disseminated sclerosis in which most marked improvement has taken place:—

**CASE 5. TYPICAL DISSEMINATED SCLEROSIS: MARKED IMPROVEMENT.**—Male, aged 19, single, a labourer, was admitted to the Edinburgh Royal Infirmary on 13th July 1901, complaining of difficulty in walking, giddiness, etc.

**PREVIOUS HISTORY.**—The patient states that he has had two attacks of rheumatic fever, the first when 15, the second when 16 years of age; he also had an attack of influenza two years ago (at the age of 17). His present illness commenced eighteen months ago—when he was 17½ years old—the first symptoms were pain in the back, headache, giddiness and double vision. About a year ago he began to complain of weakness in the legs. Eight months ago his legs became very shaky. The weakness and shaking in the legs have gradually increased; his walking has been so unsteady that during the past three months he has been frequently accused of being the worse for drink. During the last three months he has noticed that his left hand and arm have become much weaker and that he cannot lift things steadily.

**STATE.**—A well-nourished, muscular man; expression very vacant and silly-looking. Gait extremely unsteady, ataxic and spastic; left leg markedly weak, right leg slightly weak; left arm weaker than right; dynamometer—right hand = 120, left hand = 70. All the deep reflexes, both in the upper and lower extremities, are markedly exaggerated; knee-clonus present in both legs, especially the left; ankle-clonus present on both sides, more marked on the left; plantar reflex shows double extensor response. Marked nystagmus; some speech affection (articulatory defect); slight tremor of the hands; some volitional tremor of the head. Pupils dilated, 7 and 8 mm. respectively, active to light and accommodation; occasional diplopia; no dimness of vision; no optic atrophy. Some delay at times in commencing the act of urination; bladder and bowels otherwise normal. Complains of a prickling feeling (pins and needles) in the fingers and toes, but there are no objective

derangements of sensation. Complains of pain in the lower part of the back. No mental symptoms. The patient was discharged from Hospital on 20th September 1901, *in statu quo*.

SUBSEQUENT PROGRESS.—Readmitted to E.R.I. on 11th October 1901, *in statu quo*. Sent to Convalescent Home on 22nd November.

After this, marked and rapid improvement occurred.

CONDITION IN JANUARY 1905.—Patient states that for the past three years he has been employed as a van-driver and has never been off work a single day. He has lost the vacant expression of countenance; he walks well, but is still, he says, "a wee bit stiff"; close observation shows slight unsteadiness in gait; the knee-jerks are still markedly exaggerated; ankle-clonus and an extensor response still present on both sides; very slight nystagmus is still present. In every other respect the patient appears to be perfectly well.

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### SOME ASPECTS OF ALCOHOLISM.

By A. HILL BUCHAN, M.A., M.B., M.R.C.P.E.

(Continued from p. 111.)

*Seasonal Incidence* will be considered separately.

*Treatment*.—The general plan of treatment adopted was as follows. On admission the patient, when considered in a fit condition, was put in a warm bath. This was frequently found to have a beneficial effect in quieting excitement and thus obtaining a better vantage ground on which to commence drug treatment. On removal from the bath the patient was put to bed, the temperature and pulse recorded, and a full examination of the patient made in every instance. Chloral Hydrate gr. xx and Bromide of Sodium gr. xxx were then administered four-hourly till sleep was obtained. For the following figures regarding the effect of these drugs I am indebted to Dr Bruce. Mild cases slept for from 1 to 8 hours after one such dose of chloral and bromide, and on the third night slept without any hypnotic. More acute cases slept after two or more single doses. About 20 per cent. of cases required double doses. Exceedingly few patients did not respond to chloral and bromide.

These drugs were selected for routine treatment in the ward after trial had been made of practically all commonly used hypnotics. They were found safe and reliable. No bad effects

were observed to follow their use. They did not appear to set up gastro-intestinal disturbance and were well retained. In some cases where motor excitement was specially marked, hypodermic injections of hyoscine were found to be of value.

Whenever the patients were violent or manifested signs of the likelihood of their becoming dangerous to themselves or others, their movements were restrained with wrist and ankle straps. Objections have frequently been raised to this method. But it is difficult to see what other plan can, with safety to the patient and others, be adopted in hospital practice. To trust to "moral" suasion is out of the question in any case at all acute. The suddenness with which dangerous symptoms appear is a feature of the disease and must always be kept in mind in directing treatment. But even when a sufficient relay of attendants can be obtained, it may fairly be questioned whether a patient in the acute stage of the disease is likely to be more excited by mechanical restraint than by the presence of a couple of attendants whom he is apt to suspect of harbouring a desire to injure him. In every instance the restraint was removed at the earliest possible moment consistent with safety to the patient. Alcohol was not administered in uncomplicated cases.

The diet consisted chiefly of milk and soups, administered warm and at intervals of 2 to 4 hours.

As already mentioned, 93·38 per cent. of cases treated in the main on the above line recovered. It is interesting to compare these figures with older mortality statistics. Laycock examined the records of 481 cases in the Edinburgh Royal Infirmary during 11½ years. He found that of those treated with stimulants and opium, 123 or 26·7 per cent. died, while 24 cases treated without opium all recovered.

In regard to the question of the administration of alcohol in cases of D.T., it must be borne in mind that in the records we have been considering one was dealing only with cases of "medical" D.T., and that practically all the patients came into hospital with their system saturated with alcohol. From such a series it is obviously unjustifiable to dogmatise as to what is best in this respect in cases of "surgical" D.T.—cases where symptoms resembling those of D.T. appear in chronic alcoholic subjects after an injury or surgical procedure.



## II. CASES OF ALCOHOLIC INSANITY OF MORE ACUTE FORM.

A sharp dividing line cannot be drawn between this disease and D.T. and some other conditions arising in the course of chronic alcoholism.

In the more typical cases the symptom-complex is somewhat as follows:—The onset of the disease may be similar to that of D.T., but the hallucinations are not so varied and changeful, while those of an auditory character are more frequently in evidence. The true delusional element is more prominent and more systematised than in D.T. The patient reasons more about the content of his thought. Loss of orientation is less. Fear, melancholy and ideas of persecution are more marked and more difficult to dispel, the risk of suicidal attempt being consequently greater. The duration of the disease varies from a few days to several weeks, or months, recovery being more gradual than in D.T.; or it may pass into a more chronic form.

There were 48 cases which more or less distinctly conformed to this type; 37 of these were men and 11 women. Thus the proportion of women is higher in this disease than in D.T.

*Age.*—

		Male.	Female.
Average age of all cases	.	39·8	36·6 years.
„ cases which recovered		39·0	33·1 „
„ which were sent to asylum		41·7	41·4 „

TABLE XXII. SHOWS DISTRIBUTION ACCORDING TO AGE.

Age.	20-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61-65	66-70	80
Men . . .	1	4	7	8	5	5	1	1	...	1	1
Women . . .	...	2	6	5	2	2	...	...	...	...	...

*Occupation.*—(a) *Male Cases*: 1. Those specially connected with spirit trade: publicans, 2; worker in brewery, 1. 2. Other occupations: carter, 1; clerks, 2; commercial traveller, 1; confectioner, 1; mason, 1; medical student, 1; plasterer, 1; shoemaker, 1; street vendor, 1; tinsmith, 1; tobacconist, 1; transcriber, 1. (b) *Female Cases*: housewives, 2; laundress, 1.

The great majority of the patients in the ward belong to the artisan class; a circumstance which prevents one using the statistics in comparing the effects of alcohol on brain workers and manual workers.

*Heredity.*—Curiously, in only one case is there a note of alcoholic heredity. This was in the case of a man whose mother was alcoholic.

*The form in which alcohol was taken.*—Whisky in 6 cases; whisky and beer in 4 cases; whisky, beer and porter in 1 case; whisky and porter in 1 case; “port” (?) in 1 case.

*Quantity of alcohol taken daily.*—Whisky, 1 pint to 1 quart; 1½ quarts whisky; 5 glasses whisky; 3 to 4 pints whisky.

*Duration of alcoholism previous to attack.*—Of the men, 1 had taken alcohol for 6 months; 1 for 2; 2 for 4; 3 for 14; 1 for 15; 1 for 20; 1 for 33; 1 for 37 years; 6 “for years.” Of the women, one had taken it for 8; 1 for 20 years; 2 “for years.”

TABLE XXIII. SHOWING DURATION OF BOUT PRECEDING ATTACK.

Duration . . .	DAYS.			WEEKS.					
	5	8	10	3	4	5	6	7	8
Number of cases .	1	3	2	3	3	1	2	2	2

The average age at which patients began to drink was 23·9 years; the average duration of alcoholic habits 13·1 years.

*Highest Temperatures.*—

98—99 . . .	20 cases.
99·1—100 . . .	15 „
100·1—101 . . .	4 „
101·1—102 . . .	3 „
102·1—103 . . .	1 case.
103·1—104 . . .	3 cases.
104·1—107 . . .	1 case.

*Circulatory System.*—The average highest pulse rate was 98. The average lowest pulse rate was 74.

*Alimentary System.*—Nothing special noted.

*Urinary System.*—Albumen was noted as present in urine in 5 cases.

*Integumentary System.*—Excessive perspiration was specially noted in 2 cases.

*Nervous System.*—

TABLE XXIV. MENTAL SYMPTOMS.

<i>Symptom.</i>	Number of Cases which recovered.	Cases sent to Asylum.
Hallucinations noted as visual . . .	3	4
"    "    auditory . . .	3	...
"    "    auditory and visual . . .	6	4
"    "    of animals . . .	4	7
Delusions specially systematised . . .	8	6
Explanatory delusions . . .	3	...
Occupation delirium . . .	4	...
Fear specially marked . . .	4	6
Ideas of persecution . . .	13	7
Suicidal tendencies . . .	6	3
Joyful emotions . . .	1	...
Delusions taking on religious nature . . .	...	1
Impairment of memory specially marked		
(a) for recent past . . .	2	4
(b) for distant past . . .	1	4
Loss of orientation for time . . .	1	2
"    "    place . . .	4	3
Mental Symptoms persisting after sleep . . .	8	10

One patient on his recovery wrote out an account of the mental experiences he had had during his illness. The apparent recollection of detail was remarkable.

*Result.*—Twenty-one cases were removed to asylum, the rest recovered.

### III. CASES OF DOUBTFUL CLASSIFICATION.

Regarding 55 cases of alcoholism, it was found difficult to decide in what category they should be placed. (This is exclusive

of cases of simple drunkenness admitted to the ward for a night or so.)

Some of the leading points in these 55 cases were briefly as follows:—

*Sex.*—36 were men; 19 women. Average age of men, 43·3 years; of women, 36·6 years.

TABLE XXV. DISTRIBUTION ACCORDING TO AGE.

Years.	20-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61-65	66-70	71-75	76
Men . .	1	3	5	3	3	8	2	1	...	...	1	1
Women . .	2	2	3	1	3	2	...	1	...	...	...	...

*Occupations.*—Beer bottler, 1; butler, 1; clerks, 3; commercial travellers, 3; cricketers, 6; dairymen, 7; glass-blowers, 2; housewives, 9; plumber, 1; stablemen, 2; sweep, 1; washerwoman, 1.

TABLE XXVI. AGE AT WHICH PATIENTS BEGAN TO DRINK.

Years.	14-20	21-25	26-30	31-35	36-40	41-50
Men . .	3	2	1	2	1	1
Women . .	3	1	1	...	...	...

The average age of commencement of drinking was 22·1 years for the men, and 19·8 for the women.

*Average duration of alcoholic habits* in men 11·8 years.  
 " " in women 13·6 years.

*Duration of last drinking bout in weeks.*—

8 weeks	.	.	2 cases.
4 "	.	.	1 case.
3 "	.	.	3 cases.
2 "	.	.	1 case.
1 "	.	.	1 case.
less than 1 "	.	.	3 cases.

*Form in which alcohol was taken.—*

Whisky . . . . .	7 cases.
Whisky, sherry, porter	1 case.
Beer . . . . .	1 „

*Alimentary System—*

Constipation noted in	1 case.
Diarrhœa „	2 cases.
Nausea „	3 „
Pain „	3 „
Tympanitis „	1 case.
Vomiting „	2 cases.

*Urinary System.—*Albumen present in 6 cases.

*Mental Symptoms* were in most cases ill-defined, or notes not sufficient to afford clear idea of their nature.

The following table summarises them as far as could be done :—

	Cases which recovered	Sent to Asylum.	Died.
Hallucinations . . . . .	7	1	...
„ visual in character	7	1	2
„ auditory . . . . .	1	1	...
„ of animals . . . . .	1	1	...
Delusions . . . . .	7	6	2
Delusions systematised . . . . .	3	1	...
Occupation delirium . . . . .	2	...	...
Suicidal tendencies . . . . .	8	2	1
Excitement marked . . . . .	8	2	5
Orientation for time and place impaired . . . . .	4	3	2
Stupor or coma . . . . .	5	0	3
Persistence of mental symptoms after sleep . . . . .	3	6	...

*Result.*—42 patients recovered, 7 were sent to asylum, 6 died.

There were in addition to the above, 7 cases of alcoholic convulsions, in which the mental symptoms were indefinite, 4 of these being men and 3 women. The average age of these cases was 35.1 years; that at which drinking habits commenced, 29.8 years; while the average duration of alcoholic habits was 9.8 years. In 2 of them whisky, in 1 beer was drunk. The highest temperatures were 98.8, 99.2, 99.4, 102.4, 102.4, 104.4 respectively; the highest pulses, 84, 92, 100, 104, 112, 128.

Neuritis was present in 3 of these cases.

One patient, a man aged 37, had been drinking for 4 years, sometimes taking as much as a quart and a half of whisky and a gallon of beer a day. He had for 4 months been subject to fits, which only occurred after a drinking bout. When the fit was coming on he saw lights and heard bands playing. He bit his tongue while in the fit. No fit occurred during his stay in hospital.

Another case was that of a woman who had had fits for two years previously. On the present occasion five fits occurred before and three after her admission to hospital. For some days thereafter she was very confused mentally.

Another man, aged 35, in whom there was no history of previous convulsions, had been drinking heavily for three days. The day after admission he suddenly had a fit, accompanied with a cry. The arms and legs twitched, but not the face. The fit passed off in a few minutes. The patient had no recollection afterwards of its occurrence.

Another man, aged 46, had been a heavy drinker for three years, and during that time had suffered from fits, which came on in connection with drinking only. He had been drinking twelve glasses of whisky, with beer in addition, daily for six weeks, and had a fit fourteen days before admission.

Table XXVII. shows the percentage occurrence arranged according to sex and result, of some of the leading mental phenomena in all the above alcoholic cases taken together, whether D.T. or alcoholic insanity, etc. These diseases having a common cause, it may be permissible to compare individual symptoms in this way.

TABLE XXVII.

	MALE				FEMALE			
	Recovered	Sent to Asylum	Died	Total	Recovered	Sent to Asylum	Died	Total
Illusions . . . . .	3.4	0.3	0.6	4.2	2.9	...	...	2.9
Hallucinations . . . . .	28.8	2	2	32.8	28.5	5.8	5.8	40.1
Hallucinations without delusions . . . . .	16.8	1.1	0.6	18.5	1.4	1.4	1.4	4.2
Visual hallucinations . . . . .	14.2	1.2	0.2	15.6	8.8	...	1.5	10.3
Auditory hallucinations . . . . .	5.7	...	...	5.7	5.8	1.4	...	7.2
Visual and auditory hallucinations . . . . .	8.5	0.8	0.6	9.9	5.8	2.9	2.9	10.6
Hallucinations of animals . . . . .	22.8	1.1	0.6	24.5	8.8	2.9	...	11.7
Occupation delirium . . . . .	5.1	0.8	0.3	6.2	...	...	...	...
Systematised delusions . . . . .	4.4	2	0.3	6.7	1.4	7.5	...	8.9
Explanatory delusions . . . . .	1.4	0.3	...	1.7	...	1.4	...	1.4
Fear specially prominent . . . . .	4.2	0.6	...	4.8	1.4	...	...	1.4
Depression . . . . .	0.3	...	...	0.3	...	...	...	...
Ideas of persecution . . . . .	12	1.7	0.3	14.2	16.1	7.5	1.4	15.0
Suicidal tendencies . . . . .	3.4	0.3	0.6	4.3	2.9	...	...	2.9
Excitement specially prominent . . . . .	22.8	2.5	3.7	29.0	26.4	7.5	7.5	41.4
Impairment of memory . . . . .	3.9	0.8	...	4.7	10.2	2.9	1.4	14.8
Do. specially noted for								
(a) recent past . . . . .	3.1	0.8	...	3.9	8.8	2.9	1.4	13.1
(b) remote past . . . . .	0.3	0.6	...	0.9	...	1.4	...	1.4
Impairment orientation for								
(a) time . . . . .	3.7	1.7	0.8	6.2	1.4	2.9	...	4.3
(b) place . . . . .	4.5	1.1	0.6	6.3	1.4	7.5	...	8.9
Persistence of mental symptoms after sleep . . . . .	9.7	2.8	1.4	13.9	2.9	7.5	1.4	11.8

More especially with a view to seeing in how many of the cases sent to asylum alcoholism was the essential ætiological factor, and in how many it was symptomatic or merely an accidental accompaniment of some other neurosis, inquiry was made into the after-history of as many of the cases as possible. I have to express my best thanks to Dr Clouston and to Dr Douglas MacRae for most kindly furnishing notes on this point. Of course only those cases in the ward in which alcoholism was present have entered into the present series; and on comparing Dr MacRae's notes with the records, it will be seen that the majority of those diagnosed while in the ward as cases of alcoholic insanity were when under further observation in the asylum also regarded as belonging to that category.

In 8 cases, however, this was not so, and one must look on the alcohol element in these as having been incidental.

Taking these 8 cases first, we have here indicated briefly

(1) the more salient mental features noted while they were in the ward, and (2) their after-course in asylum.

Case 1. Male. Had been drinking 7-8 glasses whisky a day. *In Ward*, Sept. 1894: tremor, neuritis symptoms, strabismus, memory and orientation affected. *In Asylum*: general paralysis. Died July 1896.

2. Male, age 57. *In Ward*, Sept. 1894: violent, occupation delirium, dysarthria. *In Asylum*: general paralysis. Died April 1896.

3. Male, age 33. Had been drinking for 15 years. *In Ward*, Oct. 1894: neuritis symptoms, tremor tongue, excitement, abnormal religious emotions, delusions of possessing much money. *In Asylum*: general paralysis. Died Aug. 1896.

4. Female, age 50. *In Ward*, Jan. 1896: wild excitement, many delusions of religious nature. *In Asylum*: simple melancholia (climacteric insanity). Recovered.

5. Female, age 25. *In Ward*, Oct. 1897: excitement, fear, visual and auditory hallucinations, delusions, incoherence, impaired memory. *In Asylum*: stuporose melancholia, insanity of pregnancy. Labour, March 1898. Recovered June 1899.

6. Female. *In Ward*, March 1898: incoherence, abnormally emotional, impairment memory. *In Asylum*: general paralysis.

7. Male, age 34. Drinking for 7 years. *In Ward*, Aug. 1900: violent excitement, visual hallucinations. *In Asylum*: syphilitic insanity. Recovered Oct. 1900.

8. Female. *In Ward*, June 1900: abnormally emotional, auditory hallucinations, delusions. *In Asylum*: delusional mania + dementia.

On examining in a similar fashion the 21 cases in which the diagnosis of alcohol being the essential cause of the patients' mental condition was substantiated in the asylum, we find the following:—

Case 1. Female, age 60. *In Ward*, Oct. 1894: excitement, incoherence, marked mental confusion, symptoms persisted after sleep. *In Asylum*: alcoholic brain disease. Died Oct. 1894.

2. Male, age 27. *In Ward*: auditory hallucinations, systematised delusions, ideas of persecution, suicidal tendencies, excitement, persistence of symptoms after sleep. *In Asylum*: acute mania, alcoholic insanity. Relieved.

3. Female, age 60. *In Ward*: hallucinations of animals,



ideas of persecution, systematised delusions, excitement, melancholy, persistence of symptoms after sleep. *In Asylum*: simple mania, alcoholic insanity. Recovered.

4. Male, age 34. *In Ward*, Sept. 1897: hallucinations, visual and auditory, hallucination of animals, occupation delirium, systematised delusions, fear, persistence of symptoms after sleep. Recovered Dec. 1897.

5. Female, age 41. *In Ward*, Sept. 1897: systematised delusions, ideas of persecution, impaired orientation. *In Asylum*: delusional melancholia, alcoholic insanity. Recovered March 1899.

6. Female, age 57. *In Ward*, Dec. 1897: hallucinations, delusions about stomach being full of animals, ideas of persecution, systematised delusions, persistence of symptoms after sleep. *In Asylum*: 6th attack melancholia, delusional alcoholic insanity. Recovered Jan. 1898: has not returned.

7. Female, age 51. *In Ward*: visual and auditory hallucinations, impairment of memory and orientation. *In Asylum*: simple mania, alcoholic insanity. Died, "Brain disease" and malignant disease.

8. Male, age 64. *In Ward*, April 1898: excitement, hallucination of animals, etc. *In Asylum*: simple melancholia, alcoholic insanity. Recovered July 1898.

9. Male, age 31. *In Ward*, July 1898: visual hallucinations, hallucination of animals, systematised delusions, ideas of persecution. *In Asylum*: alcoholic insanity. Recovered Sept. 1898.

10. Female, age 27. *In Ward*, Aug. 1898: delusions, great fear, excitement. *In Asylum*: delusional melancholia, alcoholic insanity. Recovered Dec. 1898.

11. Male, age 49. *In Ward*, Oct. 1898: excitement, incoherence, impaired orientation, stuporose. *In Asylum*: stuporose melancholia, alcoholic insanity. Recovered Dec. 1898.

12. Male, age 48. *In Ward*, Nov. 1898: delusions, ideas of persecution, excitement. *In Asylum*: simple mania, alcoholic insanity. Recovered March 1899.

13. Female, age 41. *In Ward*, Jan. 1899: hallucinations, excitement, impairment of memory, especially for near past. *In Asylum*: acute mania, alcoholic insanity. Recovered Nov. 1899.

14. Male, age 40. *In Ward*, Dec. 1898: systematised

delusions, ideas of persecution. *In Asylum* : simple mania, alcoholic insanity. Recovered Jan. 1899.

15. Male, age 54. *In Ward*, Dec. 1898 : hallucination of animals, delusions, incoherent talk, excitement. *In Asylum* : simple mania, alcoholic insanity. Recovered Jan. 1899.

16. Male, age 41. *In Ward*, Feb. 1899 : illusions, visual hallucinations, delusions, ideas of persecution, excitement. *In Asylum* : delusional mania, alcoholic insanity. Recovered March 1899.

17. Male, age 63. *In Ward*, July 1899 : comatose, later violent. *In Asylum* : simple mania, alcoholic insanity. Relieved.

18. Male, age 36. *In Ward*, Oct. 1899 : visual hallucinations, hallucination of animals, occupation delirium, systematised delusions, ideas of persecution, excitement. *In Asylum* : simple mania, alcoholic insanity. Recovered Dec. 1899.

19. Female, age 61. *In Ward*, Aug. 1899 : violent excitement, visual hallucination of animals. *In Asylum* : delusional mania, alcoholic insanity. Still in Asylum, Feb. 1905.

20. Male, age 61. *In Ward*, Nov. 1899 : stuporose. *In Asylum* : simple mania, alcoholic insanity. Recovered.

21. Male, age 39. *In Ward*, Sept. 1900 : delusions of religious nature, delusions of persecution, at times excited. *In Asylum* : simple mania, alcoholic insanity. Recovered Oct. 1900. Seen in 1904 in state bordering on D.T.

The above shows the prognosis of alcoholic insanity as more hopeful than might at first sight appear from a consideration of the Ward statistics taken by themselves. Thus out of 21 cases of true alcoholic insanity, in which a reliable after-history was obtained, 15 recovered. Only one died, and that case was complicated by malignant disease. The remainder were "relieved" or are still under treatment.

The following analysis of cases of alcoholic pneumonia in the Ward under the care of Dr Bruce was made by Dr Elizabeth Erskine :—

From October 1st, 1893, to March 31st, 1899, the cases of simple uncomplicated lobar pneumonia in alcoholic subjects were 31 in number. Catarrhal and hypostatic pneumonias have not been included.

Among these the lesion in 20 was at the base.

6   "   "   apex.

3   "   "   apex and base.

Of the remaining 3 cases there were no notes.

Right base was affected in	.	5
Left " " "	.	9
Both bases were affected in	.	6
Left apex . . . .	}	2
Right base . . . .		
Right apex . . . .	}	1
Left base . . . .		
Left apex (only) . . . .	.	3
Right " " . . . .	.	3

All these were cases of simple uncomplicated alcoholic pneumonia. Nine recovered.

It was found that the number of recoveries in the pneumonia cases increased when alcohol was used in their treatment.

### **A NOTE ON NERVOUS LESIONS PRODUCED MECHANICALLY BY ATHEROMATOUS ARTERIES.**

By G. ELLIOT SMITH, Cairo.

IN the literature relating to the pathology of the brain, I think that sufficient attention has not been paid to the rôle which is played by diseased arteries in the mechanical production of lesions.

During the last twelve months I have seen no less than six cases in the dissecting-rooms of the Cairo School of Medicine and the post-mortem-room of the Kasr-el-Ainy Hospital, in which lesions of the brain—in one case leading to a fatal result—have been caused in a purely mechanical way by the pressure exerted by atheromatous arteries.

My attention was first called to this matter by a case which occurred in the dissecting-room here at the beginning of last year. The subject was a Turk about sixty years of age, whose cranial cavity was a veritable museum of morphological and pathological curiosities. The pacchionian bodies were distributed along enormously enlarged middle meningeal veins, which formed on each side a large "lac sanguin" of the dura mater: this was lodged in a very deep furrow (about 1 cm. wide)

PLATE 11.



Photograph of the medulla oblongata distorted by pressure of atheromatous arteries.  
Note the left carotid artery beginning to press on the optic nerve.



alongside the coronal suture; and the bone forming the floor of this furrow was so thin that when the man fell, as the result of a large cerebral hæmorrhage, the skull fractured along the whole course of both middle meningeal veins. These veins were the chief efferent venous channels from the brain, the superior longitudinal sinus being greatly reduced in size and importance. There were no pacchionian bodies in the usual situation; the chief collection of these bodies was found just in front of the left inferior precentral sulcus.

There was an aberrant circumolivary pyramidal bundle, such as I described in this *Review* last year, and many other anatomical peculiarities.

But the most surprising feature of the case was the complete atrophy of the left and a partial atrophy of the right optic nerves, caused by the pressure of enlarged atheromatous internal carotid arteries in the neighbourhood of the anterior clinoid processes. Since then I have seen four more cases of partial optic atrophy caused by the pressure of the atheromatous internal carotid.

I sought in vain in books and journals dealing with cerebral and ophthalmic pathology for any reference to such a cause of optic atrophy, which did not appear to be known to any of the ophthalmic physicians whom I consulted. My friend Dr Harold Nolan has, however, just called my attention to the following reference in the second American edition (1903) of Ernest Fuch's "Text-Book of Ophthalmology": "In old people a low degree of non-inflammatory atrophy of the optic nerve sometimes occurs, caused by atheromatous disease of the internal carotid, or of the ophthalmic artery. In this case the vessels, by the pressure they cause, induce a partial atrophy of the optic nerve, which for a certain part of their course they directly adjoin (Bernheimer, Sachs, Otto)," p. 493.

As I have seen no less than five cases in one year, and have had so much difficulty in finding any record of this presumably common causal factor in producing nerve lesions, it seemed worth placing it on record once more.

In my sixth case, atheromatous disease of the upper parts of the vertebral arteries, and the lower part of the basilar artery, compressed and distorted the medulla oblongata, and probably was the cause of death. The patient was brought into hospital

in a moribund condition, with a history of paraplegia and ill-defined respiratory and intestinal troubles. When the body was dissected in the anatomical department, there was nothing to explain these symptoms but an extreme distortion of the medulla oblongata. As the result of atheromatous disease, the upper ends of the two vertebral arteries had developed a large bend backward and toward the right side. These arteries had pressed upon the pyramids and pushed them to the right, and the whole medulla oblongata had become flattened and distorted.

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## Abstracts

### ANATOMY.

#### THE EXTRA-CELLULAR PATHS OF NERVOUS CONDUCTION.

(67) (*Su le vie di conduzione nervosa extra-cellulari.*) O. FRAGNITO,  
*Annali di Neurologia*, 1904, f. v.

IN this paper, with which, at the recent congress of the *Società Freniatrica Italiana* held at Genoa, he contributed to the opening of a discussion upon the anatomy and physiology of the extra- and endo-cellular paths of nervous conduction (the subject of the endo-cellular paths being introduced by Donaggio), Fragnito considers the present position of the above question. After passing in review the histological and experimental observations upon which the various opinions regarding it have been based, he formulates the following four conclusions:—

1. The paths of extra-cellular nervous conduction cannot be regarded, from the point of view of their origin, as appendages of the nerve cells; histological and experimental researches agree in showing that they are genetically autonomous.

2. The existence of fibres, which, when fully developed, terminate, according to Nissl's hypothesis, freely at either extremity without contracting direct relations with nerve cells, has not been demonstrated with equal clearness.

3. If we may regard the existence of the so-called *neuropil*—a kind of extra-cellular network formed by the anastomosis of neurofibrils derived from various nervous elements—as demonstrated in invertebrates, we cannot do so as regards vertebrates.

4. The question of the relation of one nervous element to another in fully developed vertebrates is still very obscure.

Judging from the analogy of what appears to have been established for invertebrates, we should be led to believe that the relation is one of continuity. Definite proof cannot, however, be said to have yet been furnished.

W. FORD ROBERTSON.

**ON THE THALAMUS.** GUSTAV MANN, *Brit. Med. Journ.*, Feb. 11, (68) 1905, p. 289 (17 figures).

THE central nervous system consists of a thalamic and a post-thalamic portion. These are separated from one another by a plane passing through the posterior commissure. Three stages in the evolution of the thalamus may be distinguished: primarily it was a sensory mechanism subserving smell and sight and, possessing no motor-mechanism of its own, it employed the red nucleus of the post-thalamic region as a motor-centre; secondarily it received tactile impressions from the cord or post-thalamic region and developed centres for the co-ordination of smell, sight and touch; finally it developed its own motor-mechanism in the cerebrum. Thus the thalamus of mammals includes the corpus striatum (caudate, lenticular and amygdaloid nuclei), the claustrum and the cerebral hemispheres; while the post-thalamus includes the corpora quadrigemina, the cerebellum, the pons, the medulla and the cord. The sense of smell is in all animals originally the most essential sense, as rabbits, for example, with both olfactory bulbs removed, succumb owing to their inability of realising the presence of food. Attempts to educate the senses of taste and of sight to act vicariously have so far failed. Rabbits with only one olfactory bulb removed or after the enucleation of both eyes grow up in good health. In mammals the thalamus appears as two oblong masses, which meet one another in the middle line at almost a right angle: each mass may be divided into a dorsal and a ventral portion, these being separated from one another by the falciform nucleus of Cajal, which corresponds to the winglike projection of Nissl's "nucleus of the middle line." The surface of each mass looking towards the middle line may be called the "ventricular" third and the lateral aspect of the thalamus the "lateral" third; between these two thirds is the "sub-ventricular" third. The central portion of the thalamus lies between its right and left halves in the middle line and corresponds to the ventral grey matter and to the ganglion habenulæ. Each of these thirds is divided from before backwards into an anterior, a middle and a posterior portion.

The thalamus is composed of the following nuclei:—

1. The anterior nucleus. It consists of two portions: a large-celled dorsal ventricular (Nissl's anterior nucleus; v. Monakow's



*ant. c.*; Cajal's angular nucleus [?]) and a small-celled ventral sub-ventricular portion (Nissl's antero-ventral nucleus; v. Monakow's *ant. a.*; Cajal's dorsal nucleus).

2. The ventricular nucleus. It is bounded externally by the internal medullary lamina and internally by the central grey matter. Antero-dorsally it passes into the nucleus magnocellularis of Nissl, while its posterior portion corresponds to the median centre of Luys (Nissl's mesial-posterior nucleus; v. Monakow's *med. b.*). Here, again, a division into a small-celled and a large-celled portion is quite distinct. The portion next the central grey matter is v. Monakow's *med. a.*

3. The lateral nucleus. It lies between the internal and the external medullary laminae. Its most posterior portion is in contact with the pulvinar and represents Flechsig's "semilunar nucleus." Its anterior portion is redivisible into anterior, middle and posterior parts which correspond to Nissl's "lateral-anterior" and "lateral-posterior" nuclei and to v. Monakow's *lat. a.* and *lat. b.*

4. The posterior nucleus. It is divisible into a more mesial and into a more lateral or pulvinar portion. The pulvinar is much more developed in the monkey than in the rabbit.

5. The ganglion habenulæ or epithalamus (Edinger). It is composed of a small-celled ventricular and a large-celled sub-ventricular part. It is comparatively feebly developed in the monkey, while it is large in the rabbit.

6. The external geniculate body. It is composed of a dorsal and a ventral sub-nucleus.

7. The internal geniculate body. It consists of an anterior and a posterior portion.

8. The pineal body.

9. The retinae.

10. The reticular nuclei. They lie immediately external to the external medullary laminae and are composed of three distinct sub-nuclei in the marmoset monkey, the most posterior of which subserves sight (external geniculate bodies), while the middle one is developed in connection with hearing (internal geniculate bodies). The most anterior segment seems to be developed in connection with the lateral nucleus (No. 3). Till the exact function of these nuclei has been determined experimentally it is best to classify them under a separate heading.

All the above nuclei lie in the dorsal half of the thalamus, while the ventral half contains:—

11. The ventral nucleus. It shows a distinct division into three zones. In it terminate the fillet fibres.

In the original paper are 17 figures, 14 of which represent different horizontal planes through the brain of the marmoset monkey.

AUTHOR'S ABSTRACT.

- ON THE ORIGIN OF THE EPIPHYSIS CEREBRI AS A BILATERAL  
 (69) LATERAL STRUCTURE IN BIRDS AND AMPHIBIA,  
 AND ITS INNERVATION THROUGHOUT THE VERTEBRATA. JOHN CAMERON *Proc. of Royal Soc. of Edin.*, 1902-1903 and 1903-1904; also *Journ. of Anat. and Physiol.*, April 1904; *Proc. of the Scot. Micros. Soc.*, Vol. iv., No. 1, 1904.

SOME observations which the author has made both in the chick and in the embryo frog on this somewhat enigmatical structure, tend to show that it arises in the very earliest stages of its development as a bilateral outgrowth from the roof of the thalamencephalon in these Vertebrate types. Of these two primary epiphysial elements, the one situated to the right of the mesial plane appears to become suppressed, or to blend with the more vigorously developing left outgrowth at an early period, and, as a consequence of this, the epiphysial opening in the roof of the thalamencephalon is at first situated slightly to the left of the mesial plane. This condition of matters lasts for only a very short time, as with the continued progress of development this opening tends to become situated more or less in the mesial plane, so that the early bilateral condition is in this way very soon masked, and ultimately obliterated.

These primary bilateral epiphysial recesses in Amphibia and Birds correspond to the right and left epiphysial outgrowths described by Béraneck in Lacertilia, Hill in Teleosteans, Locy in Elasmobranchs, and by Gaskell in *Ammocetes*. The results of the present research are, however, best appreciated when compared with those of Dendy in *Hatteria*; for this observer shows that the pineal eye in this animal is developed from the *left* epiphysial evagination.

The author has not yet had an opportunity of extending these researches to Mammalian embryos, but from a study of the superior commissure throughout the Vertebrata (*Journ. of Anat. and Phys.*, April 1904), he has been enabled to obtain remarkable collateral evidence which tends to point to the bilateral mode of origin of the epiphysis in this class of Vertebrates also. This minute commissure, which arises from cell-elements in the ganglia habenulæ, he found in embryos of *Zoarces* (a Teleostean) to consist firstly, of fibres which course between the right and left ganglia; and secondly, of fibres which pass to the epiphysial outgrowth of the opposite side—these latter fibres thus forming a decussation, or, in other words, a commissure or *chiasma*.

In *Iguána*, de Klinckowström finds that the pineal eye is innervated from the *right* ganglion habenulæ, so that, in this case also, the fibres perhaps cross over from right to left. This has, however, not been definitely proved in this animal, for it is not yet

known with certainty if the pineal eye in *Iguána* is really derived from the left epiphysial outgrowth.

The most interesting results were obtained, however, in the case of the adult human brain; for a study of the commissura habenularum (the posterior fibres of which were found to be homologous with the superior commissure of lower Vertebrates) by means of the Weigert-Pal method, distinctly showed that there are in existence fibres which arise from one ganglion habenulæ, and cross over to supply the opposite half of the pineal body—the fibres from both ganglia thus forming a chiasma. Thus in *man*, there is an arrangement exactly similar to that of those fibres of the superior commissure which innervate the right and left epiphysial elements of *Zoarces*, an observation which points strongly to the fact that the human pineal body is also a bilateral structure possessing a bilateral nerve supply.

The Invertebrate nature of the pineal eye has been pointed out by de Graaf in *Anguis*, Spencer in *Hatteria*, and by Gaskell in *Ammocetes*. The latter observer has also shown that the ganglia habenulæ of *Ammocetes* furnish the nerves for the pineal eyes of this animal. He therefore entitles them the optic ganglia for these eyes, and compares them with the optic ganglia found in connection with the median eyes of Arthropods. So also in the higher Vertebrates the ganglia habenulæ may be designated the optic ganglia for the pineal structures, and it may now be noted that the innervation of these is also arranged on the Invertebrate plan, for the nerve fibres have been shown to take origin in the cells of the ganglia habenulæ, while their peripheral terminations are in the pineal structures.

There can now be little doubt entertained regarding the fact that the Vertebrate epiphysis was a *paired* ancestral structure, possessing a bilateral nerve supply. It is further of great interest to note that evidence of the existence of these innervating fibres can still be found in the adult condition of *man*—the most highly evolved of all the Vertebrata.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

**AN EXPERIMENTAL STUDY OF THE RELATION OF THE  
(70) NERVOUS SYSTEM TO THE DEVELOPING MUSCULA-  
TURE IN THE EMBRYO OF THE FROG.** R. G. HARRISON,  
*Amer. Journ. of Anat.*, 1904, Vol. iii., p. 197 (18 text figures).

THIS valuable contribution begins with a succinct review of the basis for the opinions held by previous investigators. The author shows that Neuman and Herbst, each reviewing the same material

in 1901, came to different conclusions; the former held that the development of muscle takes place under the influence of the nervous system and through the agency of motor nerves; that after the muscles have arisen, their further nurture takes place independently of the nervous system and that only in post-embryonic life is the relationship re-established, as evidenced by trophic changes in the muscles removed from the influence of motor nerves. Herbst concluded that the sensory nerves and ganglia are necessary to stimulate the differentiation of muscular substance.

Harrison determines to add by experimentation to the hitherto insufficient evidence, and he further enlarges the scope of the problem by determining whether normal functioning is also necessary for structural and functional differentiation.

The experiments were conducted upon embryos of several species of *Rana*. At a stage before there was a trace of histological differentiation of nerve and muscle tissue, the whole central nervous system from the region of the pronephros to the tail was cut away, and the embryos allowed to develop. Study of serial sections abundantly proves the power of the axial musculature to develop without nervous influence. The study of the possibilities in the formation of complete limbs with normally arranged muscles has not been completed.

In the second portion of the paper the influence of the nervous system is removed chemically by the use of acetone-chloroform (chlorotone) in dilute solution. By this means it is possible to abolish all, even reflex, movements. Here, again, the embryos show that differentiation of musculature takes place in a normal manner, although all possibility of functional activity was removed by anæsthetisation of the nerve centres. On being placed in water, the embryos were able in a few moments to conduct themselves like the controls. This review may well close with the author's concluding sentence: "It must nevertheless be emphasised that all the constructive processes involved in the production and specific function of muscle fibres, and of functional activity of the muscle itself, take place independently of stimuli from the nervous system and of the functional activity of the muscle itself."

G. V. RUSK.

**ON THE TEACHING OF FLECHSIG WITH REGARD TO THE  
(71) PERCEPTIVE AND THE ASSOCIATIVE ZONES. A Paper  
read before the International Medical Congress of Madrid.  
L. BIANCHI, 1903.**

THIS paper is an enquiry into the truth of the doctrine of the physiological significance of the associative zones as enumerated

by Flechsig. Flechsig appears to suggest that each of the embryological cortical territories of the three groups—primary, intermediary, and tertiary—has a distinct physiological character, and also that these territories form the basis of so many specific forms of mental activity. These territories he has distinguished by observing the myelinisation of the intra-hemispherical fibres during the development of the brain.

But the phenomena of anatomical evolution cannot correspond to those of the development of functions. You may find the myelinisation of all the bundles going to the parietal region and to the supposed centre for reading complete in the brain of an imbecile who has never learned to read; and, moreover, it is doubtful whether the myelinisation of the bundles follows a constant law and a constant sequence.

The author then discusses the statement of Flechsig that the centres for the memory are distinct from the centres of perception of objects. If this statement were true, and if, for example, the occipito-parietal zone in front of the visual zone was found to have an associative function, receiving, as Flechsig admits, perceptions furnished by the auditory, olfactory and tactile, as well as by the visual sensory zones, which go to build up the psychic components of the highest order, then Flechsig's hypothesis might be considered probable. But this zone is concerned almost entirely with the visual function.

In man the anterior terminal extremity of this zone is exclusively visual, and is destined to the function of forming and preserving images of graphic signs of words.

All this large zone, called by Flechsig the zone of association, is in reality connected with the visual function in all its grades, *i.e.* from the simple luminous perception around the calcarine fissure, the cuneus, and the occipital pole, through the addition of the oculo-motor elements for the formation of the images of objects, and on to the formation of visual graphic signs of the same objects and their relations, in the neighbourhood of the anterior limits of this zone.

If, therefore, bilateral lesions of this zone give rise not only to a psychic blindness for objects, but also to a state of dementia more or less grave, the dementia must express a loss of a large part of patrimony of the human intellect, which has been formed from the visual images of the external world.

Nor is Fleschig's doctrine supported by histological analysis. The zones of perception should be more simple in structure than the associative centres; but histological facts do not support this contention.

Bianchi suggests that this question can best be settled by facts connected with the zones of language.

We know that word deafness is associated with destruction of the middle and posterior part of the first temporal convolution, and that a profound dementia is the result of such a lesion. This region corresponds to the 7th primary and the 23rd intermediary territories of Flechsig. Hence we find a high intellectual function, localised in these primary and intermediary territories.

Now, the centre for auditory verbal images synthetises not only auditory and kinesthetic elements of words, but also the visual, tactile and muscular images formed in the other zones, in so far as they refer to the object expressed by the word. It is therefore the centre of a very high intellectual function.

The author next dealt with the motor zone. Although it is considered a zone of projection, it is easy to understand that it receives nervous stimuli from the various sensory zones which take part in the psychic reflexes, which put us into relation with the external world. If such be its function, it should be considered an associative zone with more reason than the postero-inferior zone of Flechsig. But it is the first to be myelinised according to Flechsig's scheme, No. 1 primary territory.

In front of the motor zone, and forming part of it, is another zone, which reaches to the foot of the first frontal convolution and which constitutes, according to Bianchi, an extension of the motor zone which is associated with the higher complex movements of the upper limb, *e.g.* in writing. In the same way, the foot of the third frontal convolution, which is part of the motor zone of articulate language, is situated just in front of the zone for the more simple movements of the lips, face, tongue and larynx. But these motor centres for writing and articulate speech are important parts of the highest intellectual functions, and must contain large numbers of associative fibres.

But Flechsig himself has changed the number of cortical territories in his various publications. In 1898 he mentioned 40 territories, and at Turin 36. Moreover, his results do not coincide with those of Vogt.

According to the author, the most important fact which Flechsig has established is that all the cortical zones do not receive their fibres of projection at the same time or in equal number.

But the point about which the most important discussion is centred is the extent of the zones of perception.

Bianchi states that it is a mistake to consider *only the field of distribution of the thalamo-cortical optic fibres as the zone of perceptive vision*. This is the zone of luminous perception; but for the visual perception of objects, other coefficients are employed and a larger field is interested. This larger field is the true visual perceptive zone, and is evidently not a simple zone of projection, but is an associative zone.

Flechsigs has admitted in his last work that every sensory zone possesses in its corona radiata both sensory fibres and centrifugal fibres, probably motor, and there can be little doubt that the sensory zones, as understood in the larger sense, are zones of association.

The number of fibres of projection diminishes towards the periphery of the zone, and here the fibres of association are most numerous; while, however, the peripheral zone is more associative than the primary sensory zone, it still remains perceptive and mnemonic.

The zones which we call associative are really fields which are developed functionally by education. An example of this may be seen in the inferior parietal lobe. Lesions of this area in the uneducated only disturb the intellectual processes to a small degree; but in a patient who has been educated and who has read much, lesion of this area leads to the loss of a large part of the intellectual patrimony, a true visual aphasic dementia.

Cases are cited by the author to support these statements, and to show that lesions of the same area can produce results which are extraordinarily different in the illiterate, and in people who have done much reading. Support is also given to these views by the fact that lesions of the same portion of the right parietal lobe do not produce these intellectual disturbances, excepting in left-handed people.

While images of objects are formed bilaterally, and injury to Flechsigs's visual perceptive zone on one side of the brain does not impair the intelligence to any great degree, because the other side of the brain can carry on the work, visual images of the words which describe these objects are formed only on the left side, and therefore destruction of the centre for words on this side is followed by much disturbance of intelligence in those people who have acquired much of their intelligence by reading.

One part of this large zone of Flechsigs is perceptive for the graphic symbols of words, and it is also associative, because a large association of the products of other sensory zones is necessary to understand the verbal signs. The doctrine of Flechsigs, that the records of images are reproduced by the action of the associative centres without the intervention of the centres of perceptive sensibility, cannot be maintained. Moreover, it is not in agreement with the law that every nervous organ gives that which it produces, and reproduces that which it has formed and preserved, and which it has given.

Bianchi quotes a case in support of this view, and also shows that a study of microcephalic brains would lead to important results.

In conclusion, he offers the opinion that the only region of the

brain which can be considered to be purely associative, is the pre-frontal zone, *i.e.* a zone in which there are no fibres of projection, but which is the field in which all the elaborated products of the perceptive zones are fused together. R. G. Rows.

### **PATHOLOGY.**

**THE PATHOLOGY OF INFANTILE PARALYSIS (ACUTE (72) ANTERIOR POLIOMYELITIS).** BATTEN, *Brain*, Autumn 1904, p. 376.

THREE cases are recorded with the object of showing that the condition, contrary to current opinion, is directly due to thrombosis or embolism.

The view is usually advocated that there occurs a primary inflammation, succeeded by a thrombosis of vessels. Batten argues rather that it is primarily occasioned by a thrombosis of vessels, followed by inflammatory changes and hæmorrhage. Experimentally the occlusion of spinal cord vessels has never been followed by inflammatory change or hæmorrhage; the reply is that obliteration of a large vessel is by no means necessarily comparable to the condition which is produced when thrombosis occurs in smaller vessels. By means of fine tobacco seeds, infarcts can be produced in various organs, in which experiment, congestion, hæmorrhages, and exudation of cells were the early results of obliteration of small vessels.

And, again, when we remember how closely the area of softening corresponds to the distribution of a branch of the anterior median artery, it is but reasonable to suggest the great probability that such a condition is due to an occlusion of a vessel or vessels. The disease is more likely to occur in the lumbar region, owing to the blood supply of this portion of the cord being at a point most distal from the heart, and the long course of the reinforcing arteries.

S. A. K. WILSON.

**REPAIR OF THE NEUROFIBRILS AFTER SECTION OF THE (73) HYPOGLOSSAL NERVE.** (*Sur la réparation des neuro-fibrilles après la section du nerf hypoglosse.*) MARINESCO, *Rev. Neurol.*, Jan. 15, 1905, p. 5.

THE cells of the hypoglossal nucleus are of the reticular type, *i.e.* the neurofibrils of the cell processes penetrate the cytoplasm, ramify, anastomose and form a superficial and deep net-work.

Twenty-nine days after nerve section, the cells are atrophied



or undergoing repair. In the altered cells the appearance of the neurofibrils may be arranged into four groups. (1) Atrophied cells showing only the granular debris of neurofibrils. These cells as a rule have no prolongations. (2) Cells containing a network less well indicated than in normal cells. This is sometimes retracted, sometimes granular. (3) Cells showing neurofibrils interlacing, and here and there a network. (4) Cells with a striated appearance, in which the neurofibrils run parallel to each other; more often, however, they interlace.

By the forty-eighth day all the above appearances are more obvious. The striation is more marked, the neurofibrils are hypertrophied, and some cells show a reticular structure, especially around the nucleus.

By the sixty-second day the cells appear to have diminished a little in volume, and there is a marked tendency towards reticulation, especially in the central and posterior cells of the nucleus.

At the end of one hundred days the reparative process has advanced considerably. The deep network is formed at the centre first. During reconstruction of the network, the neurofibrils have a reticulo-striate appearance.

The neurofibrils of the protoplasmic processes are increased in size, are directed towards the cell centre, where they are lost in the perinuclear network. In their course they give off collaterals.

After section of the hypoglossal, in the reactive and reparative phase, the neurofibrils have a striate arrangement. The regenerated network differs from the original one, in that its strands are not so regular nor so fine. They are thicker and more opaque. This hypertrophy of the neurofibrils appears to be the essential element of reconstruction.

After section of the sciatic nerve, the small obscure cells of the spinal ganglia show reparative changes by the fourteenth day. In both large clear and obscure cells the fundamental substance is deeply stained, and the fibrillar network granular. The strands are thickened and the meshes enlarged and elongated. The regeneration of the network is best seen around the nucleus.

In both spinal ganglia and motor cells, in this phase of reaction, there is pallor, granularity of the neurofibrils, and more or less deep staining of the fundamental substance. In the majority of spinal ganglion cell types and in the hypoglossal, the modification of the neurofibrils, in reaction and repair, begins around the nucleus.

DAVID ORR.

**PSYCHOLOGY.**

**REACTION-TISSUES AS A TEST OF MENTAL ABILITY.** GUY  
(74) MONTROSE WHIPPLE, *Am. Journ. of Psych.*, Oct. 1904, p. 488.

THIS paper is an attempt to show: (1) that a distinction must be made between what may be called the "laboratory" and the "anthropometric" types of reaction experiment; (2) that many of the reaction tests conducted upon school children have not conformed to the usual requirements of "laboratory" method, and that reaction-time tests, of whatever type, cannot be successfully used as tests of school children, and fail to indicate mental ability.

The author criticises the work of Bagley, who attempted to correlate mental and motor ability in school children; of Gilbert, who tested school children of various ages, using a visual discrimination reaction, and also a simple visual reaction; and of Wissler, who applied various physical and mental tests to students of Columbia University as compared with one another and with class standings.

The conclusion is reached that the constant individual differences between groups of children, which have been assumed in some studies to indicate differing grades of mental ability and general intelligence, are largely products of faulty experimental conditions, and cannot, in any event, be referred, either in theory or practice, to constant individual differences in mental ability.

The outcome of any psycho-physical test does not depend merely upon the objective conditions of the test, but is influenced to an appreciable degree by the ability of each child to understand and carry out the instructions. "When, therefore, a test is affected in this way, any assumed correlation between the quantitative results and the general intelligence of the group of children tested is, in reality, but a correlation of general intelligence with itself."

W. B. DRUMMOND.

**CLINICAL NEUROLOGY.**

**THE DISEASES OF THE NERVOUS SYSTEM DUE TO EXHAUSTION.** (Die Aufbrauchkrankheiten des Nervensystems.)  
(75) L. EDINGER, *Deutsch. med. Wochenschr.*, Nos. 45, 49, 52, 1904; Nos. 1 and 4, 1905.

THIS paper is an attempt at the further elaboration and wider application of the theory the author propounded ten years ago. According to it, various diseases of the nervous system may result when there is not sufficient nutritive compensation of the katabolic loss which results from function. The anatomical basis of this conception is the axiom proposed by Weigert, that the cells

of a tissue are normally in a state of equilibrium, and that when the one is weakened or diseased, its neighbours proliferate and crush it out. Such weakness may result from a relative or absolute excess of function, and there is considerable evidence that this factor alone may result in complete degeneration of nervous element, that under certain conditions work may lead to destruction of a tract or of cells. The conditions are—(1) Abnormally great stress of work, even when the facility for recovery of the resulting loss is good; as occupation-atrophy, occupation-neuritis. (2) When there is not sufficient power of recovery, even though the function is not excessive. This generally depends on the presence of some noxious agent, as lead, syphilis. As examples are, multiple neuritis, tabes, combined system degeneration, general paralysis of the insane. (3) When there is a developmental defect of some portion of the nervous system, which has consequently not sufficient power of resistance, even to normal function, *e.g.* the hereditary nervous diseases—Friedreich's ataxy, spastic paresis—amyotrophic lateral sclerosis, non-tabetic optic atrophy.

Under this conception the form which the disease takes does not depend directly on the noxious agent, but on the effect of the stress of function of parts which are ill-nourished, where there is a relative deficiency in nutrition, or where the power of resistance is subnormal. Thus function determines the form and distribution of the paralysis. As examples, the various forms of lead palsy may be cited; in painters the extensors of the fingers and wrists are chiefly affected, while the supinator longus, a little-used muscle, generally escapes; in compositors the small hand muscles often suffer most; and in file-cutters, according to Moebius, the thumb-muscles.

The author's suggestive views on the nature and origin of tabes dorsalis according to this hypothesis are well known, and he adds little to them in this paper.

The various points of his arguments are abundantly supported and illustrated by the citation of clinical cases of his own observation and from the literature of the subject.

GORDON HOLMES.

#### **A CASE OF ACUTE ANTERIOR POLIO-MYELITIS IN AN ADULT,**

(76) **WITH CENTRAL FOCAL LESIONS.** (*Un cas de polio-myélite antérieure aiguë de l'adulte avec lésions médullaires en foyers.*) A. LÉRI and S. A. K. WILSON, *Nouv. Icon. de la Salpêtr.*, Nov. 6, 1904.

FOR a long time past, abundant clinical evidence has been forthcoming to demonstrate that acute anterior polio-myelitis is not a

disease confined to children, but the number of cases in adults which have been followed to the post-mortem room has been very small. In this article the authors record in detail the clinical manifestations of such a case during the acute attack, which was observed in 1895 in the Bicêtre under the care of M. Pierre Marie, and notes are given of the further history of the case until death, which occurred in 1903 from tuberculosis.

The man was 23 years of age, and had contracted a chancre 5 years before. After some ten days of preliminary sharp pains in the right leg, and to a less extent in the left, an attack of fever, with vomiting and diarrhoea came on, suggestive of typhoid fever. On the third day of the fever he noticed that he could not move the left leg, and that the right was weak: at this stage he was admitted to hospital. It was then found that the left knee-jerk was absent, and that the right was very weak: there was general cutaneous hyperæsthesia, but no anæsthesias. The paralysis continued to increase for about a week after the onset of the fever, the upper extremities being affected on the fifth day. There was transient albuminuria for about a fortnight.

The attack then subsided, and recovery began to occur. After tenotomy, he recovered sufficiently to enable him to get about without assistance.

At the autopsy, there was found a double focal lesion which had destroyed symmetrically but not equally the greater part of the grey matter of the anterior horns of the cervical and lumbar enlargements. These patches resembled exactly the foci of infantile paralysis. In the main, a blood-vessel was present in the centre of each patch, whilst around the vessels were much dilated and surrounded by large open perivascular spaces. The appearances suggest to the authors that the lesion is primarily a vascular one—either thrombosis or embolism of the arteries of the anterior horns—and probably of syphilitic origin.

STANLEY BARNES.

**TWO CASES OF FAMILIAL HEREDO-SPINAL ATROPHY (FRIED-  
(77) REICH'S TYPE) WITH ONE AUTOPSY, AND ONE CASE  
OF SO-CALLED ABORTIVE FORM OF FRIEDREICH'S  
DISEASE. Parts II. and III. MINGAZZINI and PERUSINI,  
*Journ. of Ment. Path.*, Vol. vi., 1904, p. 57.**

THIS instalment of the paper contains the results of a careful histological examination of the material obtained from the case in which an autopsy was performed, and a discussion of the essential clinical features of the disease and its relations to Marie's cerebellar ataxia.

The specimens were prepared in the usual way, with fixation by chromic salts. Hence no important cytological observations were possible, and the examination was restricted for the most part to determining the topography of the lesion and the coarser changes induced by the disease.

The pia mater was found to be thickened along the entire extent of the cord, as were also the septa which pass from it into the substance of the cord itself. These were distinguished from neuroglial tissue by their intense red colouring with van Gieson's stain. The blood-vessels entering the cord were observed to be thickened, and the change in them as in the pia and septa seemed to diminish in intensity in passing from the lumbar to the cervical segments of the cord.

The alterations noted in the substance of the cord itself correspond with what is known from other cases. The most advanced degeneration was found in the posterior columns, and especially in that part which eventually forms the column of Goll, whilst the posterior roots were "appreciably degenerated," as was also Lissauer's zone.

In the antero-lateral columns a diffuse marginal degeneration existed in the periphery of the entire column, and seemed to be in direct relation to the thickened pia. The crossed pyramidal tract, the direct cerebellar tract, and part of Gower's column were also altered in varying degrees.

In the grey matter the endo-cornual network was impaired, especially in the posterior cornua, whose cells, moreover, appeared to be poorly stained and diminished in number. Clarke's column was disintegrated, neither cells nor fibrous network being observed.

The "tourbillons" described by Déjerine were present, and were very abundant in the parts where the destruction of fibres was most advanced.

Tufts of interstitial tissue fibrils were found in the periphery of the spinal cord, especially in the lumbar region. These were immediately contiguous with the pia mater and seemed to irradiate from a central nucleus, but the question of whether they arose from the pia or from neuroglial elements was not decided. In the lower medulla the degenerations of the various tracts gradually disappeared, and above that level no morbid changes were found. The cerebellum was absolutely intact.

In discussing the clinical features of the disease, Soca's synopsis is accepted as a fairly satisfactory summary of the symptomatology, but the existence of numerous atypical cases is freely conceded.

The most interesting points in this connection are naturally the relationship of Friedreich's disease to Marie's cerebellar ataxia, and after referring to the views of various writers on the subject, the

authors conclude, in agreement with Amouroux, Baumlein and others, that "there are two types of familial ataxia—Friedreich's and Marie's type. Between these two there are intermediary varieties." Further discussion of this statement they reserve for the section of their paper on "Physio-Pathology," and this section has not yet been published. Clinically, however, they point out that if the differential diagnosis is based on the presence of ocular disturbances, the conservation or exaggeration of the patellar reflexes, and the tardy onset of the disease, many cases will be found to present great difficulty in being referred definitely to one or other of the two types.

HARRY RAINY.

**PITUITARY TUMOUR WITHOUT ACROMEGALY. (Hypophysealstumoren ohne Akromegalie.)** JENÖ KOLLARITS, *Deut. Zeitsch. f. Nervenheilk.*, Bd. xxviii., Heft 1, S. 88.

AFTER referring to Marie's view that disease of the pituitary gland is the cause of acromegaly, Kollarits discusses the question whether the disease is caused by increased, lessened, or altered secretion, or is due to its entire absence. Before giving details of his two cases, he gives a brief review of the literature of the subject, referring especially to the theory of Brissaud and Meige that acromegaly and gigantism are one and the same disease, the difference in result merely depending upon whether the epiphyseal cartilages of the patient have become ossified or not; while Cestan and Halberstadt carry this theory still further, and suggest that in later life, when periosteal bone formation has become less active or is entirely absent, acromegaly and thickening of the bones do not supervene, the specific symptom now being obesity.

The author tabulates a set of over fifty cases of hypophyseal disease without acromegaly, collected from the literature of the subject; and from a study of these he concludes that disease of the pituitary is not the cause, but merely one of the symptoms of acromegaly. He then describes two fatal cases, the one of a girl, aged 17, with sarcoma of the pituitary spreading to the bones of the base of the skull; and the other that of a woman of 28, with angio-sarcoma of the gland spreading into the cranial cavity and nares, and compressing the base of the brain. In neither of these was acromegaly present.

W. E. CARNEGIE DICKSON.

**AFFECTIONS OF THE FACULTIES OF READING, SPEECH AND**

(79) **WRITING IN GENERAL PARALYSIS.** (Des troubles de la lecture, de la parole, et de l'écriture, chez les paralitiques généraux.) JOFFROY, *Nouv. Icon. de la Salpêtrière*, Nov.-Dec. 1904, p. 409.

As an introduction to an interesting clinical lecture, Professor Joffroy defends his differentiation of the difficulties in writing encountered among general paralytics into "caligraphic" and "psychographic" disorders. From the study of a paralytic's writing we may gather useful indications of his mental status. Changes in the form of the writing reveal a retrogression in an acquired faculty: alteration of its content, the graphic "exteriorisation" of thought, is still more valuable as a sign of psychic deterioration.

Similarly, one may distinguish "arthrolalic" and "psycholalic" difficulties in speech; "arthrolexic" and "psycholexic" difficulties in reading.

As far as reading is concerned, general paralytics furnish a classic demonstration of arthrolexic disorders, characterised for an instance by slowness, stammering, slurring, syllabic reduplication; and so on. As psycholexic impairment one may indicate the intonation, so often not in agreement with the thought expressed in the sentence read by the patient: some trifling fact receives the emphasis: an amusing tale is read in a sorrowful tone of voice. Ask him what he has just read: either he will have no idea of it, or at the most will have retained a confused and contradictory notion. No unfailing comparison, however, is to be drawn between the duration of the disease and the degree of the arthrolexic or psycholexic deterioration.

A similar series of pathological phenomena is to be noted in the domain of speech. Arthrolalic impairment is of course highly typical of general paralysis, and it is very commonly associated with tremulousness of the face and tongue, "a veritable dance of muscle fibres."

More interesting are the psycholalic characteristics of the disease. It will be sufficient to enumerate inaccuracy in the choice of expressions, a sort of "psychic inco-ordination" leading to this inaccuracy, and transitory motor and sensory aphasias.

For the writing of the general paralytic it is often difficult to make a definite distinction between the two varieties of phenomena. Thus the fact that his writing is often laboriously slow may be explained by defective functioning of his muscular system or of his higher centres. If he is excited, he writes quickly; if depressed, slowly. His letters and words are irregular, uneven, separated; further, the impersonal element in his caligraphy, the result of

education, disappears, and it becomes more natural, then infantile or childish. Similarly it must be remarked that there is no definite relation between the duration of the disease and the degree of alteration in the handwriting.

S. A. K. WILSON.

#### CONTRIBUTION TO THE SYMPTOMATOLOGY OF HEMIPLEGIA.

(80) (Zur Symptomatologie der Hemiplegie.) K. HEILBRONNER,  
*Deutsche Ztschr. f. Nervenheilk.*, Bd. 28, 1904, Heft 1, S. 1.

HEILBRONNER calls attention to a phenomenon which is normally present in the ordinary dead body (after *rigor mortis* has passed off) if left spread out on a horizontal surface. It also occurs during life in cases of recent hemiplegia, on the paralysed side. The phenomenon, which he calls "broad leg," consists in an outward rotation of the lower limb at the hip-joint (analogous to what occurs in fracture of the neck of the femur), due to the weight of the soft parts. The diameter of the limb is thus apparently decreased from before backwards, and the whole thigh appears broadened. In recent hemiplegia the contrast with the normal side is striking. Thus, whilst the circumference of the non-paralysed thigh is more or less circular, that of the paralysed side is a flattened oval. In hemiplegia this phenomenon is easily demonstrated when in a sitting posture on a firm seat. At first sight one might think that the affair is simply due to external rotation of the hip. But if the paralysed limb be passively extended by pulling on the foot and then rotated inwards till the bones on the two sides are parallel, there is an "overflow" of the soft tissues of the paralysed thigh, encroaching on the sound side. This is due to flaccidity of the muscles and soft tissues, which roll about as in a loose bag. If the thigh be passively lifted up, the mass of tissues hangs like an oval, with its long axis from before backwards.

Similar appearances are also seen in other muscular masses, *e.g.* those of the calf and upper arm. These form loose bags of tissue, which flatten out under the influence of gravity. Muscular patients with scanty subcutaneous fat show these phenomena well, as also do patients with firm, compact, subcutaneous fat. If the fat be loose and flabby, as in old female cases, or in patients previously fat who have subsequently emaciated, the phenomenon is less distinct, since such patients already have bags of fat loosely surrounding the limb. But, even then, the sitting posture will usually demonstrate the difference between the two sides. In bilateral paralysis the diagnostic significance is lost, owing to the want of a sound limb for comparison.

This phenomenon, according to Heilbronner, does not occur in



the flaccidity of normal sleep, since even during sleep the muscles are not completely relaxed. During sleep there is usually no respiratory flapping of the lips, as in hemiplegia. Nor does the unconsciousness of deep narcosis from anæsthetics produce "broad leg." But he has observed it in cases of severe acute polyneuritis and in recent poliomyelitis before the onset of atrophy. In tabes the phenomenon is less distinct; moreover, in this disease we do not have a sound limb with which to compare, and there is always the additional possibility of abnormal laxity of the ligaments in tabetic limbs.

The phenomenon does not occur in hysterical hemiplegia, nor in simulated paralysis.

Heilbronner regards *breites Bein* as an evidence of the hypotonia resulting from diminished sensation of resistance on passive movement, with consequent possibility of extreme excursion of the joints.

The phenomenon is of immediate onset, and sometimes it has lasted for as long as eight weeks, long after the knee-jerk had returned, and was even increased, and with appearance of ankle-clonus. It may even persist for some time after voluntary motor power has begun to reappear in the quadriceps extensor, but as soon as the tonus of the paralysed limb returns, the phenomenon disappears. In all cases, however, it is accompanied by diminished resistance to passive movement of the joints.

PURVES STEWART.

#### ON CERTAIN TREMORS IN ORGANIC CEREBRAL LESIONS.

(81) GORDON HOLMES, *Brain*, Autumn 1904, p. 327.

THE author condemns the loose way in which the term "tremor" has been employed in literature, and emphasises the necessity for a detailed description and graphic record of the movements of which the tremor is composed. He suggests that the term should be used to denote "a clinical phenomenon consisting in the involuntary oscillation of any part of the body around any plane, such oscillations being either regular or irregular in rate and in amplitude, and due to the alternate action of groups of muscles and their antagonists."

He gives notes of nine cases which presented clinical symptoms of considerable similarity, and he draws the conclusion that the site of the lesion in each case was in the mid-brain, involving more especially its dorsal or tegmental portion, and possibly in some cases extending into the posterior part of the optic thalamus: this localisation was verified by autopsy in two cases of tumour.

One constant symptom was tremor, so similar in all the cases

that he thinks it may be regarded as peculiar to or dependent on a lesion of the dorsal or tegmental portion of the mid-brain, and therefore a sign of diagnostic importance. The tremor was of slow rate, three to five oscillations per second, and generally of large amplitude; it ceased during sleep, and also when the limb involved lay at complete rest, so supported that each of its segments was individually supported; it was increased by mental excitement, by forcible movements of the opposite limb, and, often, by voluntary attempt to check the tremor—in every case the patient was unable to check it for more than the shortest space of time; in every case, volitional movement of the limb affected was complicated by an irregularity of the intention-tremor type.

Each case presented other symptoms, (*a*) those more or less associated with the symptom complex; and (*b*) those merely casually related or coincident, but of great importance as indications of the localisation of the lesions. The former group includes slight rigidity, or, better, stiffness of limbs; “uselessness” of the affected limbs, in great part merely subjective; a peculiar emotional state, especially tendency to uncontrollable attacks of laughing or crying, mental apathy and slowness; paresis of the expressional movements of the face. The latter group includes especially ocular palsies—these were a prominent symptom in all cases except one, and loss or defect of the vertical movements was most frequent; definite hemiplegic weakness, with increased reflexes and extensor plantar response; a peculiar gait, resembling closely that either of paralysis agitans or of cerebellar disease; hemianæsthesia, always slight in degree and not of cortical type, often with marked subjective loss of feeling: in two cases, homonymous hemianopia on the side of the affected limbs, with Wernicke’s hemiopic pupil reaction.

From a consideration of these symptoms, Holmes draws the conclusion that each case of the series presented a focus of disease involving the dorsal portion of the mid-brain, and he regards it as probable that the tremor noted in all cases is a symptom peculiar to a lesion of this region. Further, he adduces arguments in favour of the view that the essential and constant factor in the lesions is involvement of the nucleus ruber, or, at least, of the cerebello-rubro-spinal system, and that the tremor is due to destruction, not irritation, of these parts, with consequent over-action of the fore-brain cortex. It must, then, be assumed that the cerebello-rubral system normally exerts an inhibitory influence on cortical action: Holmes does not find sufficient evidence to decide whether this inhibitory function is normally exerted on the higher or lower motor centres, on the cortex or on the spinal cord.

A full review of literature on disorders of movement is very difficult, as there has been but little attempt to distinguish between

or classify the various forms (chorea, athetosis, tremor, etc.): yet there is such evident difference between these varieties that "it seems warranted to rigidly separate them in clinical study, and to assume that they must be due to, or constantly associated with, lesions different in nature or site." Such symptoms have been found "practically only in disease of the region extending from the internal capsule to the upper margin of the pons." Holmes has collected from literature 60 cases, with autopsies, which showed such spontaneous involuntary movements (athetosis in 15, chorea in 20, tremor in 25), and he thinks it may be stated that athetosis almost always results from disease of the basal ganglia, especially the optic thalamus, that choreiform movements occur with about equal frequency in lesions of the basal ganglia and of the mid-brain, while in the large majority of the cases of tremor the lesion involves the midbrain. He discusses shortly the various views which have been held regarding the pathogenesis of these involuntary movements.

Attention is drawn to the remarkably close resemblance between the symptoms of some of his cases and the classical symptomatology of paralysis agitans, *e.g.* in respect of the tremor, gait, rigidity of body, mask-like expression, etc., and he suggests the possibility of the lesion of paralysis agitans being in the red nucleus, of the nature of a slow and chronic sclerosis, secondary to a vascular condition (arterio-sclerosis). A. W. MACKINTOSH.

**ON THE RELATION BETWEEN VARIOUS ATMOSPHERIC  
(82) CONDITIONS AND THE OCCURRENCE OF CEREBRAL  
HÆMORRHAGE.** RUSSELL, *Lancet*, Jan. 28, 1905, p. 222.

THE writer's conclusions are drawn from a series of cases of cerebral hæmorrhage occurring at the Birmingham General Hospital from 1885 to 1902. He considers that there seems to be a slight tendency to the occurrence of cerebral hæmorrhage on days of high atmospheric pressure and also on days of rising pressure. There is a marked tendency on days of low wind pressure, while the occurrence of low wind pressure combined with high barometric reading is the condition under which the largest number of cases take place. Apart from season, temperature itself has not been shown to exert any influence, though more cases take place during the six colder months of the year than during its warmer half.

JOHN D. COMRIE.

**THE INFLUENCE OF STIGMATA OF DEGENERATION UPON**

(83) **THE PROGNOSIS OF EPILEPSY.** WILLIAM ALDREN  
TURNER, *Med.-Chir. Transac.*, Lond., Vol. lxxxviii.; *Lancet*,  
Feb. 18, 1905.

THE purpose of the communication was to ascertain, by means of a statistical investigation, whether the presence of structural stigmata of degeneration exercised any influence, and if so, to what extent, upon the prognosis of epilepsy.

Stigmata of degeneration are defined as structural deviations from the normal, arising during the periods of development and brain growth, in those who are the subjects of a hereditary degenerative predisposition.

One hundred consecutive cases of confirmed epilepsy from amongst the patients resident at the Colony for Epileptics, Chalfont St Peter, were examined with this object.

The stigmata to which attention was especially directed were:—  
(1) Facial, including nasal deformity and asymmetry. This was found to be more common in females than in males. Although present in 42 per cent. of the total number of cases, it was only found in 14 per cent. as a single stigma. (2) Deformities of the hard palate. These form some of the most frequent and trustworthy of degenerative phenomena. Abnormal palates were found in 42 per cent. of the cases, but only in 17 per cent. as a solitary stigma. (3) Deformities of the external ears, indicated by abnormal size, shape and symmetry, were present in 33 per cent., but in only 9 per cent. as a solitary stigma. (4) Dental anomalies and displacements.

Brief reference is also made to stammering and high errors of refraction as physiological stigmata.

The statistical facts bearing upon prognosis are considered under the following headings:—

1. *Sex.*—Of 100 epileptics, 62 were males and 38 were females. Males exhibit neuropathic stigmata to a much greater extent than females, in the proportion of 52 males to 23 females. Of the total number of cases, males and females, 75 per cent. presented stigmata, and 25 per cent. were free. It is pointed out that the greater freedom of females from stigmata is in relationship to the greater exemption from mental deterioration seen in the female sex.

2. *Hereditary history.*—Owing to the difficulty in obtaining precise and trustworthy information on this point, it was not possible to deduce any general conclusions, but it was observed that direct parental heredity to epilepsy or insanity was usually associated with more pronounced stigmata than collateral heredity.

3. *Age at onset of the convulsions.*—Those cases in which the

disease commenced between birth and five years of age showed a larger percentage of stigmata in comparison to those without any than at other ages. This is in harmony with what has been elsewhere shown, that epilepsy commencing during the first quinquennium is characterised by the highest percentage of cases showing profound mental impairment, and is most favourable for the development of the confirmed malady.

4. It was apparent from a study of the *duration of the convulsions* that the absence of stigmata did not necessarily imply an early or favourable termination of the disease.

5. *The type of the seizures.*—The greatest difference in the proportion of cases, with and without stigmata, was observed in those which presented the combined grand and petit mal type. Such combination has also been shown to be associated with the highest grades of dementia. The purely petit mal type did not exhibit marked evidence of a hereditary degenerative disposition. Cases of serial epilepsy do not show marked evidence of stigmata.

6. The relation between the presence of stigmata and *the mental state* was especially obvious. Those epileptics who only showed the slighter degrees of mental impairment presented a nearly equal proportion with and without stigmata (21 per cent. with and 19 per cent. without). On the other hand, of those in whom there was marked mental enfeeblement, 53 per cent. exhibited and only 7 per cent. were free from them. Hence there would appear to be a close association between degrees of mental impairment and the presence of neuropathic stigmata, a fact which is proffered as an argument in favour of the view that the interparoxysmal mental condition in epilepsy is an integral part of the disease.

AUTHOR'S ABSTRACT.

**THE IMPORTANT CLINICAL POINTS IN PERIMETRY, WITH  
(84) SPECIAL REFERENCE TO TRAUMATIC NEUROSIS.**

L. WOLFFBERG (Breslau), *Archiv. Ophthalm.*, Vol. xxxiii., No. 6, 1904, p. 597.

IN the November 1904 issue of Knapp's *Archives of Ophthalmology*, this article appears as an abridged translation by Dr Ward A. Holden. The author, after referring to the fact that our information on this subject is very incomplete as found in literature and standard text-books, proceeds to warn workers in this subject against too easily accepting as normal any statement which may be given as to the limits of the field of vision in health. He points out how variation in facial prominences, in the size and position of the pupil, in the refraction and so on, may in varying degree modify the extent of the field of vision. In addition to

the extent in all directions, the integrity of the visual function throughout the entire area of the field must be considered.

The question of illumination during perimetric examination is dealt with, and some most important and highly interesting points are brought forward.

In 1883 and 1884, Wolffberg, while he was assistant to Sattler at Erlangen, made up a tissue-paper arrangement for the window-frame, so that the illumination of the perimeter could be varied as desired, and the amount of light in the room photometrically stated. In this way a field taken in ordinary daylight can be controlled by any degree of reduced illumination. By the use of this method some new observations were made, which are stated in a recapitulation of the important points at the end of this paper.

The above-mentioned tissue-paper apparatus has been substituted by a more convenient mode of testing the light sense. This consists of a test-card like that of Snellen, but made with blue letters on a black card. The types are such that only in bright light can  $\frac{6}{5}$  be read by the normal eye; and when the illumination is reduced so that Snellen  $\frac{6}{5}$  can just be seen, the reading of the blue letters becomes reduced to  $\frac{5}{30}$ .

When the tissue-paper shades are put in the window, the measure of the degree of illumination can be taken by recording the acuteness of vision for the blue letters on the black card.

Perimetry, in relation to the fatigue phenomenon found in neurasthenic patients, and said to occur in traumatic neurosis, is discussed. Wolffberg does not agree with Schmidt Rimpler that ordinary relaxation of attention is the cause of the fatigue type of field. Reuss was the first to furnish experimental proof that the fatigue of the neurasthenic is chiefly psychical fatigue, and not a question of simple lack of attention. If during the examination of the first eye the second be covered, so that its retina is at rest, the fatigue spiral can be elicited at once in the second eye as soon as it is uncovered.

The fatigue phenomenon can be demonstrated for central as well as for peripheral vision. Helmholtz said: "The field of vision corresponds to a drawing in which the important feature is carefully executed in detail, while the other parts are roughly sketched in, and the more roughly the farther they be from the important feature."

Hirschberg states that "the maximum acuteness of vision lies in an area no larger than 40', corresponding to the 0.2 mm. linear extent of the fovea. Over an area of from 15°-20° from the centre, colours appear saturated; while beyond this limit one colour after another fades, until the periphery appears quite grey."

The examination of the fovea is carried out by determining the

acuteness of vision in testing refraction, and by tests for the direct perception of blue and red.

The tests for the acuteness of vision in the periphery of the retina are made apart from correction of refraction. Experience has shown that even high errors of refraction do not particularly affect excentric vision. The examination of the red and blue colour sense in the field is a sufficient colour test, as in this way anomalies of the photo-chemical and neuroptic apparatus can be determined.

If a particular colour limit is to be taken as a standard, the following points must be considered:—

1. The spectral purity of the colour used. Military cloth does very well.

2. The visual angle. A patch of 15 mm. is recommended as a suitable size to use.

3. The surroundings. A lustreless deep black cloth forms the best background.

4. Adaptation of the retina.

5. Intensity of general illumination.

The first four may be regarded as constant, but the fifth is variable. The maximum limits should first be ascertained in bright daylight, and then the effect of a definite reduction of the illumination should be tested.

With his tissue paper apparatus in use, Wolffberg established the following laws:—The maximum colour limits contract when the illumination is reduced to  $\frac{1}{16}$ , gradually, but never more than  $15^\circ$  if the light sense is normal. The typical sequence, blue, red, green, is never lost. With farther reduction of the illumination the colour limits contract, and finally green disappears, then red, then blue. With a normal light sense the relation of the colour limits in the field remains constant in reduced illumination.

Notes of a case of traumatic neurosis are given. The paper concludes by a recapitulation of the more important points, as follows:—

1. The visual field that is found to be normal in bright daylight should not suffer the slightest change in limits or continuity when the illumination is reduced. This holds good up to the degree of reduction of illumination at which the fixation object ceases to be visible.

2. In every visual process of the unmoved eye three functions are concerned to a greater or less degree, namely, the dioptric, the photo-chemical, and the neuroptic.

3. The influence of the dioptric apparatus is recognised by tests of vision employed for determining the refraction.

The influence of the photo-chemical apparatus is recognised by tests in diminished illumination.

The influence of the neuroptic apparatus is recognised, on the one hand by exclusion after tests of the other functions, and on the other by a peculiar condition of the colour-sense.

4. The dioptric apparatus is of value for the macula only, since errors of refraction can be corrected practically for it alone. For examining the photo-chemical apparatus, blue pigments are best, since blue disappears first when the illumination is reduced, and since it is lost last in disturbances of the neuroptic apparatus. For examining the neuroptic apparatus, red objects are best, because the perception of red suffers quickly in cases of reduced excitability of the neuroptic apparatus, and since it is recognised better than blue in cases of disturbances of the photo-chemical apparatus.

5. What has been said regarding the influence of the three kinds of apparatus on the visual act, and their tests, holds good not only for the macula, but for any particular portion of the field, and for the field as a whole.

6. The limits of the colour field can be considered normal in a given case only when the degree of illumination is known. According to Donders and Landolt, the test is wrongly carried out if the limits of the colour field do not approach the limits for white.

7. The colour limits contract concentrically as the illumination is reduced, but if the photo-chemical and neuroptic apparatus are normal there will be no change from the normal sequence of the limits blue, red, green. If the illumination is reduced sufficiently, green disappears, then red, then blue, and colours are no longer recognised.

8. A field taken in ordinary daylight, which in its limits for white and colours corresponds to the limits obtained in the normal eye with diminished illumination, is typical of diminished excitability of the neuroptic apparatus and of abnormal fatigue, thus of traumatic neurosis.

9. Traumatic neurosis consists in an abnormal fatigue, not of the retina, but of the neuroptic apparatus, including the cerebral centres. The fatigue found in the field as a whole also exists for the macula, and is shown by the condition of quantitative perception of colour.

10. Anomalies of the photo-chemical apparatus are revealed by the relation of the blue to the red limits. The preponderance of the latter over the former may not be manifest at every point in the field, and by every degree of illumination. The perception of red exceeds that of blue in the entire field only in cases of idiopathic hemeralopia, and even in such cases the symptom can be made manifest only when the illumination is greatly reduced.

A. H. H. SINCLAIR.



**INCOMPLETE BILATERAL HEMIANOPSIA FROM GUNSHOT**

(85) **WOUND.** (Hémianopsie bilatérale avec conservation des champs visuels maculaires à la suite d'un coup de fer de la région temporale.) VAN DUYSE (Ghent), *Arch. d'Ophtal.*, Jan. 1905.

VAN DUYSE, Ghent, publishes the interesting case of a young man who, in a poaching affray, was shot in the right side of the head, on the evening of 8th December 1901. He lay senseless for an hour or two (the length of time is uncertain), and then found himself quite blind. With great difficulty he got home, where he lay for two days in hiding, but on the third day he was obliged to seek advice at hospital, when he was found to be entirely blind, but the pupils reacted (though not very actively) to light; to be perfectly sensible, and to have no motor or sensory paralysis whatever. His wound lay on the right side, 4.5 cm. above and 1 cm. behind the external auditory meatus. Into this wound a surgeon somewhat heroically passed a probe horizontally into the cranial cavity without finding any foreign body. Radiograms showed two foreign bodies; one lying behind the situation of the left auditory meatus, the other in a very similar situation on the right side. The fundus was normal, the evening temperature was slightly raised; but beyond this, the blindness, and some headache, there were no symptoms whatever. In a few days vision began to return, and eventually the state of the fields became very singular. There was an imperfect double hemianopsia, the macular vision, over a somewhat egg-shaped field, remaining intact in each eye. The loss of vision was practically complete in the left upper homonymous quadrants, and partial all the rest of the way round the circle, being worse at certain points than at others. During the next two years the patient had numerous attacks of complete loss of sight with scintillations; these were supposed to take their origin in a temporary ischæmia of the cortex from spasm of vessels; they gradually passed off, however, without permanent harm. Van Duyse found by experiments on other skulls that a projectile entering the skull where it did in the patient, and lying where it appeared to do, would certainly injure the anterior portion of the optic radiation behind the position of the lenticular nucleus. The fact of the retention of the pupillary reaction even over the blind quadrant is emphasised, and the numerous points of interest in connection with an injury so singular are carefully dealt with in the paper.

W. G. SYM.

**FUCHS' PERIPHERIC ATROPHY OF THE OPTIC NERVES.**

(86) Dr K. KIRIBUCHI, Tokio, *Arch. of Ophthal.*, Nov. 1904, p. 586.  
(Abridged Translation.)

FUCHS found that in adults there was usually an atrophy of certain bundles of fibres in the optic nerves. The affected bundles lie immediately beneath the pial sheath and also about the central vessels.

In the anterior segment of the optic nerve there are found, besides the usual longitudinal septa, those called by Fuchs peripheric septa.

Kiribuchi believes that the peripheric septa are modified longitudinal septa, which can be regarded as a portion of the pial sheath. They furnish points of attachment for the transverse septa. They are developed mainly in the anterior portion of the nerve, because there numerous transverse septa exist.

The writer has found evidences of these peripheric septa and of Fuchs' atrophy in the new-born infant and in the foetus of eight or nine months.

He regards the glia tissue between the peripheric septa and the pial sheath as nothing other than a glia mantle about the nerve, which must exist physiologically in all persons, just as the brain and cord are covered with a thick mantle of glia tissue. The glia tissue does not represent the network left after the atrophy of nerve fibres, as Fuchs believed, nor a defective development of medullary sheaths, as Michel believed.

C. H. USHER.

**CLINICAL CONTRIBUTION TO THE STUDY OF THE INNER-**

(87) **VATION OF THE IRIS.** Dr C. MAGNANI, Turin, *Ophthalmic Surgeon at Smyrna*, *Arch. of Ophthal.*, Nov. 1904, p. 591.

MAGNANI describes the case of a male, aged 20, who had received a contusion on the right eye. There was blurred vision and mydriasis. R.V. =  $\frac{20}{200}$ . Rejected all glasses. Pupil = 7 mm. Lens partially dislocated. Opacities on posterior surface of lens, but only toward the inner part. Vitreous, retina and choroid normal, with the exception of a red suffusion in the macular region.

The pupil presented no reaction to light, either directly or indirectly. It contracted on convergence, but the pupil remained always a little larger than the other, and the same difference obtained when a drop of 1 per cent. eserine was instilled, which produced contraction of the sphincter. The state of the accommodation could not be determined, owing to the defective vision and the lens opacities.

Four weeks later with -1.25 D. V. =  $\frac{20}{100}$ . The red suffusion at

the macula had almost disappeared. The pupil began to contract to light directly and indirectly.

After a prolonged period, the pupil was still a little more dilated than the other; it reacted to light directly and indirectly, and on convergence. Macula was normal. Iris tremulous. The palpebral reaction could not be produced. Closure of the lids seemed rather to produce dilatation of the pupil.

The cause of the mydriasis in this case could not be attributed to rupture of the sphincter, because the iris acted during convergence, and for the same reason the idea of paralysis of the oculomotor nerve-endings could not be entertained.

The writer considers that the contusion injured the ganglion cells described by Müller in the ciliary muscle, and therefore this form of mydriasis would be similar to the paradoxical pupillary dilatation of Langendorff, and the same that occurs in the extirpation of the superior cervical ganglion.

That the interruption was not in the afferent fibres is shown by the failure of the indirect light reaction. C. H. USHER.

#### **CONGENITAL UNILATERAL HYPERTROPHY OF THE FACE.**

(88) WERNER, *Arch. f. klin. Chirurgie*, Bd. 75, H. 2, 1904, p. 533.

In the case recorded in this paper, the enlargement involved all the tissues, both soft and bony, of the left side of the face and cranium in front of a vertical line running through the mastoid process, and including the teeth, tongue, palate, uvula and tonsil. Skiagrams showed abnormal thickness of the malar bone and base of the skull near the sella turcica. There was no difference in the activity of the salivary and cutaneous glands on the two sides, in the growth of hair, in the hearing, in the ocular functions, or in the temperature. The hemihypertrophy had maintained its relative size until recently, when (at the age of 24) it had shown some tendency to increase, so far as the size of the cheek was concerned. For cosmetic reasons a part of the hypertrophied cheek was removed. Werner does not hold to the neurotic theory (Tretat-Monod) of hemihypertrophy, but ascribes it to some abnormality of the embryonic anlage. J. S. FOWLER.

#### **ON THE CORTICAL CENTRE FOR CONTRALATERAL DEVIATION**

(89) **OF THE EYES AND HEAD.** (Ueber das Rindenzentrum für kontralaterale Augen- und Kopfdrehung.) ZUR VERTH, *Mitt. a. d. Grenzgeb. der Med. u. Chir.*, Bd. xiv., p. 195.

THE writer states that the position of the centre for the act of turning the head and eyes to the opposite side is not yet fixed with

certainty, and he records a case which he considers of extreme importance in relation to the question.

A man, aged 20, was struck in October 1902 with a hammer-like walking stick on the left side of the head. He worked for two days, and then began to be troubled by involuntary turning of the head and eyes to the right side, for which he sought advice. On examination, a round wound about the size of a sixpence was found 4 cm. to the left of the middle line, involving both scalp and skull. An operation was undertaken to remove the splinters and cleanse the wound. While the patient was in hospital, and even after the operation, he kept on having attacks of head and eye deviation, accompanied by twitching of the right side of the face, but no loss of consciousness. During 1903 these attacks gradually became more frequent, the twitching spread to the right arm and leg, and later to the left side of the body. Speech also became affected, and there was some impairment of consciousness at these times.

In September 1903 another operation was undertaken. A scar was found adherent to the edges of the dura and subjacent cortex, beneath which was a cavity containing half a teaspoonful of clear fluid. The scar was excised and the wound did well, but the patient died on the seventh day from pneumonia.

The only lesion found post-mortem was the localised scar situated in the upper part of the foot of the second frontal convolution on the left side. Two photographs in the text show its position.

The writer after discussing the subject concludes: (1) that the position of the centre for contralateral head and eye deviation lies at the foot of the second frontal convolution; (2) that the centre for the eyes is above that for the head. JOHN D. COMRIE.

**CEREBELLAR AND BULBAR SYNDROMES.** (Syndrome Cérébelloleux et Syndrome Bulbaire.) ANDRÉ THOMAS, *Revue Neurolog.*, Jan. 15, 1905, p. 16.

THIS paper records the clinical history and pathological details of the following case:—The patient was a married woman æt. 58, and the only point of interest in her antecedent history was that she had had two miscarriages, and that the only child which was born alive died shortly after birth. The clinical details were briefly as follows. In 1886 there was a gradual onset of alternating hemiplegia—paralysis of the right limbs and of the left third nerve. The hemiplegia gradually improved, but the ocular palsy persisted. In 1897 she began to have attacks of vertigo—impairment of equilibration, loss of sensibility over the distribution of the left trigeminal nerve, and intention tremor in the upper extremities,

especially the right. In 1900 she became deaf, especially on the right side. There was nystagmus and an increase in her instability, with paralysis of both lower limbs. In 1902 she developed complete paralysis of the left side of the face, and the left cornea sloughed away. She died early in 1903.

The autopsy revealed: (1) some thickening of the meninges, especially round the exit of the left third nerve; (2) a small focus of sclerosis in the upper dorsal cord; (3) a large patch in the left half of the pons and bulb lying dorsal and lateral to the lower third of the inferior olive; (4) a small focus in the lower part of the pons in the region of the central tegmental tract; (5) on the right side, softening of the upper part of the facial nucleus, and posterior to it of the superior olive. T. GRAINGER STEWART.

#### OPPENHEIM'S "FEED-REFLEX" AND CERTAIN OTHER RE-

- (91) **FLEXES.** (*Studien über den Oppenheimschen "Fressreflex" und einige andere Reflexe.*) W. FÜRNRÖHR, *Deutsche Ztschr. f. Nervenheilk.*, Bd. 27, 1904, p. 375.

FÜRNRÖHR has made observations on Oppenheim's "feed-reflex," on Henneberg's "hard-palate reflex," and on the "buccal reflex" of Escherich, Toulouse and Vurpas.

In the healthy infant, during the first few months of life, by stroking the lips, tongue, hard palate, or any part of the oral cavity, we produce a reflex, consisting of rhythmic sucking, smacking, chewing and swallowing movements, repeated from four to six times. Normally, after the age of eight to ten months this reflex is no longer present. But in certain pathological conditions it may persist, not only in older children, but even in adults. In them it may even appear spontaneously, probably the result of salivation. Oppenheim found this reflex in cases of pseudo-bulbar paralysis and of post-epileptic coma. Fürnröhr also noted its presence in post-hemiplegic coma and in the coma of hydrocephalus. In these conditions, as the coma passes off, the reflex disappears. In pseudo-bulbar paralysis it occurs without coma. The reflex is considered to be due to the uncontrolled action of a sub-cortical centre, either in the optic thalamus or more probably in the medulla oblongata. As cortical control appears in the child, this reflex disappears; but if control development be deficient, it may persist.

Henneberg's "hard-palate reflex" in healthy infants is seldom present. It is elicited by rapid, firm stroking of the hard palate from before backwards, with a rod or spatula. The result is a strong contraction of the orbicularis oris. It occurs chiefly in pathological conditions, which may be of the most varied kind, but with much less constancy than Oppenheim's "feed-reflex."

The hard-palate reflex is a short reflex muscular twitch, unlike the slow rhythmic movements of Oppenheim's phenomenon. Fürnrohr believes the two phenomena to be distinct, Henneberg's reflex being a mucous membrane reflex, analogous to the anal reflex.

The "buccal reflex" is produced by percussion of the upper lip, producing a pouting contraction of the orbicularis oris. This phenomenon, which is normally present in newly-born infants, also in anencephalic monsters, certain idiots, alcoholics, and general paralytics, is considered by Fürnrohr not to be a true reflex, but simply a sign of increased irritability to mechanical stimulation, analogous to the well-known facial irritability of tetany.

PURVES STEWART.

**MICROPSIA AND ALLIED CONDITIONS.** (Über Mikropsie und (92) verwandte Zustände.) HEILBRONNER, *Deutsch. Ztschr. f. Nervenhe.*, Bd. 27, H. 5 u. 6, Dec. 22, 1904, p. 414.

AN apparently normal twenty-seven years old man observed when at school that the book which he was reading, and the letters on the page, seemed to retreat away into the distance, although, as the letters somehow or other seemed to remain of ordinary size, he had no difficulty in continuing to read. It was only the object of his direct gaze which seemed thus to retreat: the hands which held the book did not alter in position. The phenomenon still occurs from time to time. He further suffers from attacks of giddiness, or rather, is more or less continuously conscious of feeling suddenly giddy, the sensation lasting perhaps only for a second or two. He cannot allow himself to dance as a consequence. A painter by trade, he can work only when on the ground. The mere sight of anyone on a ladder or scaffold is sufficient to cause intense giddiness. It has, however, never been noticed that he actually reels or makes any compensatory movement when thus affected.

On the other hand, all sorts of tests employed objectively to elicit this giddiness fail to produce it. Nor has he any nystagmus.

A somewhat similar case, described by Pfister, was ascribed to paralysis of accommodation, an insufficient explanation. Möbius inclines to a lesion in the calcarine fissure, to which theory also, objection may be taken. The case in point, associated as it is with a subjective state not unlike agoraphobia, is most probably due to a cortical change only, but the writer does not go further. Micropsia, or porropsia, as Heilbronner suggests (πόρρω=afar), occurs occasionally as an aura in epilepsy. Pfister's patient, when his finger was moved, felt as though it were being moved through

an enormous distance, a similar phenomenon occurring in a case recorded by Veraguth.

There would appear to be a cortical mechanism for the appreciation of the state of the body musculature generally, including the eye muscles, interference with which might lead to phenomena such as are noted in this paper.

S. A. K. WILSON.

**CLINICAL RESEARCHES ON MUSCLE SOUND.** (*Klinische Untersuchungen über den Muskelton.*) LINK, *Neurolog. Centralbl.*, Jan. 16, 1905, p. 50.

WHILE physiology has from time to time interested itself in the question of muscle sound, the references to the subject from the clinical side are meagre in the extreme. This is no doubt partly attributable to the conflict of theories as to its origin, partly to ignorance of or disbelief in its value as a means of diagnosis. Some hold it to be but a resonant tone or note of the tympanum itself; others consider it the result of a discontinuous stimulation of muscle from high level centres at a rate of about 16 to 20 impulses per second, that is during voluntary movement. No doubt tympanic vibration accompanies this muscle sound, but it appears fairly certain that the two are not identical, either in time or in rhythm.

The author uses an ordinary stethoscope, or a phonendoscope of 22 to 24 vibrations per second. Applying either lightly over a voluntarily contracting muscle, one hears unfailingly a low, deep note, which by suitable arrangement can be shown to be due neither to movement of muscle fibres nor to the coursing of the blood stream through the muscle.

In complete flaccid palsies, though the patient attempt to innervate the muscle concerned, no sound is heard. In partial paretic states it is faint, though distinguishable. During faradic stimulation of a normal muscle it is loud, and may vary in pitch according to the rate of interruption of the coil employed. If the constant current be used, there is no variation from the tone normally audible.

In the slow contraction of degeneration obtained in galvanic stimulation, no sound will be detected. The explanation, according to the author, must be found in some change (chemical or otherwise) within the muscle itself, a change evident to the eye in the slowness with which the contraction is accomplished.

During reflex contraction nothing is heard, perhaps because the duration is so short, although in the quickest possible voluntary movement the muscle note may always, or practically always, be recognised.

Not without significance is the fact that in the late contracture of hemiplegia (infantile cerebral, or adult), no sound is forthcoming, though should a voluntary movement of any affected muscle be accomplished, it is accompanied by the muscle note. In hysterical contracture, on the other hand, there is no loss.

S. A. K. WILSON.

## PSYCHIATRY.

### SOME METABOLISM STUDIES WITH SPECIAL REFERENCE

(94) **TO MENTAL DISORDERS.** FOLIN, SHAFFER and HILL,  
*Amer. Journ. Insanity*, April 1904, p. 699, and Oct. 1904, p. 299.

THE authors give the results of several years' work in the M'Lean Hospital. They call attention to the fact that the literature records no complete analysis of a 24 hours' quantity of urine, and that previous investigators in the field of mental diseases have not studied metabolism as a whole. Because of these fragmentary analyses, and the lack of any standards for comparison, much confusion has arisen, and normal variations have been falsely interpreted.

The present work is a study of the urine, and the analysis shown in the 67 tables are probably the most complete and comprehensive yet published. The value of having a "standard diet" for experimental purposes is emphasised—one that could be used generally in the same way that "test meals" are in stomach diseases. A liquid diet was used in these experiments, and the same amount given to each individual. The analytical technique is given, including some original methods.

The first series of experiments were done on eight normal persons, and in this way some values obtained for comparison. Various types of psychoses were next studied—chiefly general paralytics, manic-depressive insanity and dementia præcox.

The authors conclude that their results contain a strong suggestion that general paralysis is a disease associated with some *demonstrable* metabolism disorder; this claim is based chiefly on the great fluctuations observed; in other psychoses, while variations from the standard values are frequent, it is not possible to identify any one metabolism peculiarity with any particular form of mental disease. They believe that their researches prove untrustworthy all previous claims of characteristic findings in any of the ordinary mental disorders.

G. H. KIRBY.



**FEIGNED INSANITY: MALINGERY REVEALED BY THE USE**  
 (95) **OF ETHER.** C. WAGNER, *Amer. Journ. Insanity*, Oct. 1904,  
 p. 123.

THIS is a report of a case of some medico-legal interest. The patient was a police officer who had been convicted and sentenced to death for murder of his wife. He had been alcoholic for years; during the trial he talked incoherently and answered questions irrelevantly or not at all; he appeared unable to stand erect, and when he walked a violent coarse tremor agitated his legs. Later, during eighteen months in prison he rarely spoke, and never stood or walked without assistance. Before execution an inquiry was held into his mental condition. He then showed the same tremor of the legs, dragged his feet when assisted to walk, and when standing still bore his weight on the balls of his feet and would fall if not supported. He gave almost no attention to questions, stared vacantly, laughed and moved his lips as if whispering. Knee-jerks slightly increased, pupils normal. It was finally decided to etherise him, and just before he relaxed under the anæsthetic he became talkative and profane; when completely unconscious he was placed upon his feet, and as consciousness returned he walked with firm steps and the tremor had disappeared. After this he talked freely, admitted that he had been simulating, and had been under an awful strain. No one had told him how to act; the tremor had been suggested by a trembling of his legs which he felt one day before the trial. He remained perfectly sensible up to the time of his execution, and went to the electric chair without fear.

G. H. KIRBY.

**CLINICAL CONTRIBUTIONS TO THE QUESTION OF MELAN-**  
 (96) **CHOLIA.** (*Klinische Beiträge zur Melancholicfrage.*) O. KÖLPIN  
 (of Greifswald), *Arch. f. Psych. u. Nervenkr.*, 1904, Bd. 39, Heft 1.

KÖLPIN begins by contrasting the views of Wernicke and Kraepelin with regard to melancholia: the former looks upon "affective melancholia" characterised by a feeling of subjective insufficiency as quite different from "depressive melancholia" where retardation is a prominent symptom: "affective melancholia" is a symptom-complex which may occur alone, or in association with the anxiety-psychosis, or with the akinetic phase of a cyclic motility psychosis. Kraepelin gives the term "melancholia" to a psychosis of the involution period, and looks upon affective and depressive melancholia (which he does not distinguish) as phases of the manic-

depressive psychosis. Kölpin discusses eighteen cases of depression, and seeks on the basis of these to give a critical appreciation of these contrasted views. He first takes typical cases of affective melancholia, which he calls "pure melancholia" to avoid the association of Wernicke's term with his general views, and of the anxiety-psychosis, and then goes on to cases where the symptomatic picture is less distinct, containing features of each. The appearance of hypochondriacal ideas (cases 6, 7), and of motor symptoms are next discussed (cases 8-12). That the author uses melancholia in a rather wide sense is seen in his discussion of a case of recurrent stupor (case 9), which he calls a periodic "melancholia with catatonic symptoms." Such a case is better kept apart in the present unsatisfactory state of classification. The case of a boy aged 15 (case 11), who presented for three months a condition of akinesia, mutism, catalepsy and occasionally echopraxia, passing into a hypomanic state and recovery with some residual phenomena, is diagnosed as melancholia on what seems a very insufficient basis: the large question of the relation of hysteria to catatonia and manic-depressive insanity is mentioned, but not discussed. Two cases of catatonia (13, 14) are given in which the differential diagnosis from melancholia is discussed. Cases 15, 16 are patients with different forms of neurasthenic insanity. Case 16 is interesting, and may be put in the series of Friedmann's cases of neurasthenic melancholia.

In summing up the discussions of the various cases, the author inclines rather more to the views of Kraepelin than to those of Wernicke, although he considers the former are too extreme. Kraepelin holds that the melancholia of the involution period is quite distinct from the depressive stage of manic-depressive insanity, but he gives in his text-book no sure criterion to enable us to distinguish the two. Kölpin considers that symptomatologically it is as yet impossible in many cases to separate involution melancholia from periodic and circular melancholic conditions. He does not agree with Wernicke's strict separation of affective and depressive melancholia, but if these are both to be put under manic-depressive insanity, he insists on the importance of recognising *formes frustes* of this psychosis, which will embrace therefore not only the periodic and circular forms usually recognised, but also cases where there is a series of attacks of depression, and others where there is only a single attack. In conclusion, he insists on the importance of recognising the motor symptoms in the depressions, and refusing to consider them as peculiar to catatonia.

C. MACFIE CAMPBELL.

**ON THE DIFFERENTIAL DIAGNOSIS OF NEGATIVISTIC**

(97) **PHENOMENA.** (*Zur Differentialdiagnostik negativistischer Phänomene.*) By O. GROSS (of Graz), *Psych.-Neur. Wochenschr.*, Nr. 37, 38, 1904.

GROSS distinguishes between psycho-motor or "catatonic" negativism and the effect of aversion (*Ablehnung*): the two are often associated, and usually it is impossible to get from the patient data which enable one to determine the value of each element. A case of catatonia, who gave an excellent description of her mental condition, is reported in detail by the author to demonstrate the presence of a psycho-motor negativism without there being any effect of dislike or aversion. Patient was a girl 20 years of age, who after a short period of excitement passed into a catatonic state, with peculiar attitudes and mannerisms, hallucinations of hearing, occasional mutism and negativism. Her negativistic behaviour was accompanied by no disinclination nor aversion, which she expressly denied; in general she was compliant and co-operated conscientiously. For some days she insisted on exposing herself completely, but not in an erotic manner; also injuring herself and spitting round about her. When asked later why she had exposed herself, she said, "Because I had to; there was an inner compulsion." (Did that act on your will or your movements?) "On the will and then on the movements." She expressly denied that her movements were made against her will: "A sudden notion comes, to which I must give in; usually I feel no endeavour to resist."

As to the negativism of the patient, her unæsthetic exposure, self-inflicted injuries, and mutism, Gross says: "In this case, a complex of pathological impulses caused the repression and blocking of normal impulses and their conversion into direct opposite." Negativism is merely one phenomenon resulting from the disintegration of consciousness, which latter is due to a lowered activity of the supreme cerebral function, viz., that of co-ordinating the simultaneous activities of consciousness into one unity. When this disintegration takes place, consciousness is split up into several independent processes, one of which preserves the identity of the personality. Elements from the other side-processes may reach more than liminal intensity, however, and are then experienced as foreign to the personality, often taking the form of hallucinations, autochthonous ideas or impulses. Gross gives a resumé of the various forms of disintegration, dissociation and splitting of consciousness in the neuroses and psychoses. Disintegration of consciousness presupposes a serious disorder of the unifying principle of consciousness; this latter is aided in its effect by various factors—inner disharmonies and painful feelings. In the

disintegration of consciousness, as seen in dementia præcox, the endogenous sejunctive disposition plays the main rôle, while in the systematised dissociation of consciousness, as seen in hysteria, the exogenous factors are more important. According to Gross, the case discussed by him is a transition between disintegration and systematised splitting of consciousness.

After this rather hypothetical psychological discussion, Gross gives the case of a patient aged 49, who, after a condition of great anxiety and perplexity, passed into a chronic paranoiac state. During the condition of anxiety she often refused to co-operate, due to the painful perplexity in which she was, and which examination made more acute. In this case, therefore, the mechanism of the negativism was quite different, and only indirectly depended on the disintegration of consciousness: this latter produced a painful perplexity, felt as such by the conscious personality and translated into action as movements of aversion. In summing up, Gross suggests the following possibilities:—1. True catatonic ("psychomotor") negativism is the expression of a split-off chain of psychophysics processes not related to the conscious personality, and therefore escaping introspection. 2. The effect of aversion depends upon perplexity, which is exaggerated when patient is disturbed; it is the expression of the conscious personality. 3. "Psychic" and total negativism depends on the association of 1 and 2.

C. MACFIE CAMPBELL.

#### ON DELIRIOUS ASYMBOLY AND EPILEPTIC OLIGOPHASIA.

(98) (*Ueber delirante Asymbolie und epileptische Oligophasie.*)

A. BERNSTEIN (of Moscow), *Monatsschr. f. Psych. u. Neur.*, Nov. 1904.

BERNSTEIN calls attention to the value in certain psychoses of the simple method of making the patient name objects and pictures shown him. Asymboly or imperfect grasp of the nature of common objects, appears in the infectious-exhaustion deliria, in delirium tremens, in epileptic and hysterical confusional states, in arterio-sclerotic, paralytic and senile deliria. The author has never met with true asymboly in manic-depressive or in catatonic conditions. The author analyses the nature of the symptom in the above groups; in the infectious-exhaustion conditions it is due to hazy perception of optic stimuli, and is not characterised by the same tendency to illusions as in delirium tremens. Bernstein would look upon the reading defect of the alcoholic delirium, considered by Bonhoeffer as paraphasia, rather as asymbolic dyslexia. He suggests that in hysterical conditions the asymboly may be due to retraction of the visual field. In arterio-sclerotic and senile dementia the distract-

ability with poor attention is the basis of the asymboly. Before discussing the symptom in epileptic conditions, Bernstein gives a case of epileptic confusion where patient at 11 A.M. could not name correctly objects shown nor use them, although his general behaviour suggested nothing abnormal; at 1 A.M. patient showed that he grasped the nature of objects shown, but could not name them correctly; at 7 P.M. he could name objects and pictures shown him, save a few of the pictures. This order of recovery is the usual one.

In states of epileptic confusion there may be both an asymboly and a transitory amnesic aphasia. The latter is the longer in duration and, unless specially examined for, is apt to escape attention, because the limited number of words at patient's disposal is sufficient for his clouded mental state, whereas in aphasia with organic lesion the needs of the individual are quite out of proportion to his available speech-mechanism; Bernstein therefore uses "oligophasia" to mark this difference.

Examination of the patient demonstrates that it is a real amnesic aphasia, and that the patient, while co-operating and recognising an object, is unable to find the correct name for it; two cases of recurrent utterance are mentioned, one of ten minutes' duration. Bernstein found this temporary aphasia in twenty consecutive cases of epileptic confusion; the extent varied, the character was constant; the condition could be demonstrated up to three or four days after complete clearing of consciousness. He criticises Pick's view that the symptom is due to perseveration of ideas, and Heilbronner's explanation of it on the basis of flighty association. He has been able on several occasions to make the diagnosis of the epileptic nature of a psychosis on the basis of this symptom. The article ends with a discussion of those conditions which present disturbances that might be confused with epileptic oligophasia.

C. MACFIE CAMPBELL.

**A CASE OF GENERAL PARALYSIS AFTER TYPHOID FEVER  
(99) WITH COMPLETE RECOVERY. (Ein Fall von Dementia  
paralytica, etc.)** O. FOERSTER, *Monatsschr. f. Psych. u. Neur.*,  
Dec. 1904.

FOERSTER reports the case of a man, 42 years of age, neither syphilitic nor alcoholic, who after a severe attack of typhoid fever with delirium, presented a clinical picture similar in some respects to that of general paralysis of the insane.

Physically he showed general muscular weakness, tremor of the hands and tongue, ataxia of the lower extremities, tremor of face muscles on speaking, tremor of speech with stumbling over

syllables, tremulous writing with omission and transposition of letters, bilateral patellar clonus, no eye symptoms.

Patient's general attitude was one of indifference to his environment; he showed poor retention, ignorance of elementary school knowledge, rather hazy memory for the period of his illness; he developed some ideas of reference. On the basis of these physical and mental symptoms the author diagnosed general paralysis, and as the patient recovered he gives the above title to his communication.

The mental status as given by Foerster seems hardly typical of general paralysis; the attitude of the patient seems to have been one of apathy, no dilapidation of thought is mentioned, and from the data furnished one has little idea of the mental trend, emotional variations and general reactions of the patient. In view of the etiology and outcome of the absence of eye symptoms and of typical mental symptoms, it would only lead to confusion to call such a case general paralysis.

C. MACFIE CAMPBELL.

## TREATMENT.

**THE TREATMENT OF EXOPHTHALMIC GOITRE BY MEANS  
(100) OF ANTITHYREOIDIN.** (Ein Beitrag zur Behandlung des  
Morbus Basedowii mit Antithyreoidinserum [Moebius].)  
H. HEMPEL, *München. med. Wchnschr.*, 1905, No. 1, p. 14.  
(Einige Beobachtungen über Moebius Antithyreoidin.) K. THIENGER,  
(101) *Ibid.*, p. 15.

ANTITHYREOIDIN, or the serum of Moebius, is obtained from sheep six weeks after removal of the thyroid gland. Hempel records one case of exophthalmic goitre treated by means of that serum. The patient, a woman of 55 years, in whom cardiac symptoms predominated, was given 5 grammes of the serum by the mouth every third day for about six weeks. The exophthalmos diminished, the pulse rate fell from 140 to 96 per minute, the cardiac arrhythmia disappeared, the circumference of the neck diminished by 2 cm., the patient gained three pounds in weight, and her general condition was markedly improved.

Thienger records four cases treated by means of 5 c.c. of the serum every second day. The first case, a woman aged 25, did not improve under treatment, and had eventually to be transferred to the psychiatric wards. In the second and third cases, the main result of treatment consisted in subjective improvement of the patients' general health. The body weight was increased, and the pulse frequency lessened, but the thyroid swelling was not reduced.

Thienger's fourth case, a man aged 20, was almost *in extremis* when admitted to hospital five days after onset of symptoms, which, together with the signs of the disease, were well marked and typical. Serum-treatment was commenced on the fourth day after admission. Three days later, general improvement was observed, and the pulse rate had fallen from 140 to 96 per minute. Improvement continued steadily, and at the end of about two months, in which time 120 c.c. of serum had been administered, the patient was discharged in comparatively good health. Exophthalmos could then hardly be detected, the thyroid was soft and but slightly prominent, Moebius's and Stellwag's signs were still present, the tremor had disappeared, the pulse rate averaged 90 to 100 per minute, but the continued increase in frequency might be accounted for by the co-existing aortic incompetence.

The conclusions Thienger draws are that antithyreoidin is not a panacea for every case of exophthalmic goitre, but that, in view of the satisfactory results obtained in some cases, the serum-treatment is worthy of further trial.

W. T. RITCHIE.

**BRIDGING OF NERVE DEFECTS.** CHARLES A. POWERS, *Ann. of*  
(102) *Surgery*, Nov. 1904, p. 632.

POWERS reports a case of nerve grafting, along with a summary of a study of the literature on the bridging of nerve defects. In his own case, grafting of four inches of a dog's sciatic nerve, to replace a gap in the external popliteal nerve of a youth eighteen years old, was carried out. Eight years later sensation was partly restored, but motor paralysis remained complete, and Powers therefore classes his case as a failure.

The various methods which have been adopted to bridge defects are: grafting, as in Power's case; flap operations, carried out by turning down a flap from the central end of a divided nerve long enough to reach the distal end; implantation; resection of bone, to allow of approximation of divided nerve ends; and suture at a distance, by loops of catgut, etc. Powers has analysed the reports of twenty-two cases of nerve grafting, and states that more than 20 per cent. of these may be said to have terminated in a "satisfactory way." In spite of this he advises that nerve transplantation should be discarded. His study of the records of other methods of nerve restoration led to no more satisfactory results, for his general conclusion is that "it hardly seems possible at this time to say what form of bridging should be employed." The difficulty in formulating definite conclusions is due partly to the

fact that the reports of cases are few in number, and partly to the results attained being in many cases too vaguely reported.

J. W. STRUTHERS.

**A CASE ILLUSTRATING THE OPERATIVE TREATMENT OF  
(103) PARALYSIS OF THE SERRATUS MAGNUS BY MUSCLE  
GRAFTING. A. H. TUBBY, *Brit. Med. Journ.*, Oct. 29, 1904,  
p. 1159.**

TUBBY records a successful case of muscle transplantation for the relief of the disability and deformity following paralysis of the serratus magnus. His patient was a girl of seven years old who presented the usual features of serratus paralysis on the left side. The lesion was of uncertain origin, though probably due to anterior poliomyelitis. The operation consisted in making an incision along the anterior axillary border, freeing a portion of the pectoralis major, dividing the humeral attachment of the freed portion and reinserting it into the atrophied serratus magnus. This was done by splitting the freed end of the pectoral muscle into several fasciculi and stitching these firmly into the serratus over an area of four or five of its digitations. As the result of the operation the girl gained considerable increase in power of abducting and thrusting forward the arm and the deformity was much lessened. The power of the pectoralis major was not appreciably weakened by the operation.

J. W. STRUTHERS.



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# Review of Neurology and Psychiatry

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## Original Articles

### CAUSES AND DISTRIBUTION OF INSANITY.<sup>1</sup>

By JOHN MACPHERSON, M.D., F.R.C.P.E.

IN the two previous lectures of this course<sup>2</sup> the subject of Variation in its relation to the origin of Physical Malformations, of Congenital Mental Defect and of the Neuroses, such as Epilepsy, Hysteria and Alcoholism was considered. The correlation between congenital malformation and congenital mental defect was pointed out. The relation of the neuroses to one another, their heredity and their distribution throughout mankind of all races was insisted on. It was also shown that all these affections are genetic in origin and independent of so-called causes or influences due to the environment. For the environment is not and cannot be constant while the manifestations in question are, so far as we know, universal. It was further shown that congenital malformation and congenital mental defect are due to inherent processes, the nature of which is at present unknown, acting within the fertilised ovum. It is moreover certain that these processes must be independent of the environment of the elements contributed by either parent, of the immediate state of health of the parents or, with certain exceptions, such as injuries or special disease, of the uterine environment. It is not asserted that diseases affecting the mother or even, on rare occasions, specific affections of the father, may not deleteriously influence

<sup>1</sup> Abstracted from the Morison Lectures delivered before the Royal College of Physicians of Edinburgh, 1905.

<sup>2</sup> *Rev. of Neurol. and Psychiat.*, vol. ii., 1904, p. 285.



the growing embryo *in utero*. The fact remains, however, that in the majority of instances these defects are hereditary; that they may pass over several members of the same family, and in the case of animals over several members of the same litter—nay, even that they pass over one or more generations to reappear in a succeeding one. In face of such facts it is useless to speculate upon physical causes while the great innate cause remains obscure.

In the second lecture I discussed the distribution of the neuroses throughout the various races of mankind, but the subject was necessarily curtailed for want of time and space. It would not, I believe, be a difficult task to prove that no race of men is free from very extensive affection by epilepsy or hysteria, or many other nervous affections regarding which we have but an imperfect account. When we come to enquire into the similar prevalence of insanity, however, the evidence is much more negative, and although there is no race of men who are known to be free from it, yet on the whole travellers are in too many instances silent. In many instances it is evident that the subject does not interest them, while in still other instances they simply say that they saw no insane people. Hence has arisen the mistaken idea that insanity is a product of civilisation, and that it is rare or unknown among savage or barbarous people. I have taken the trouble to collect numerous references from the writings of travellers on this subject, and I shall read a few of them for the purpose of showing the unfounded nature of the belief which has arisen on this question. Dr Felkin informs me that he has seen in all some thirty to forty lunatics on the White Nile. He also saw some maniacs chained. He was the first to tell me a curious fact of which I have since had confirmation from other sources, viz.: that the type of insanity among the African natives is different from that in Europe. The prevailing form of mania is a short acute kind lasting only a day or two, during which the sufferer is driven away to the woods or voluntarily runs away, returning again in a few days apparently restored in mind. Idiocy was very common in his experience and so was suicide. Thompson in his book, "Through Massailand," states that he found insanity very common. The myths and folk stories of the people are full of reference to it. Those affected by lunacy are driven away from the habitations of sane

people or are otherwise isolated. He also found idiocy very common, especially among the dwarfs and albinos, the latter of whom were numerous, and about the prevalence of mental defect among them there was no doubt. In a book entitled "The Indian Tribes of the United States," edited by Francis S. Drake, reference is made to an Oregon Indian woman who appeared to be demented: "She sang in a wild manner, and would offer to the spectators all the little articles she possessed, scarifying herself in a horrid manner if any one refused to accept her presents. She seemed to be an object of pity to the Indians, who allowed her to do as she pleased."<sup>1</sup> Captain Cook in his "Voyages," referring to the South Sea Islanders, says: "We met with two instances of persons of disordered mind, the one a man at Owyhee and the other a woman at Oneheeow. It appeared from the particular attention and respect paid to them that the opinion of their being inspired by the Divinity, which obtains among most of the nations of the East, is also received here" (in the Pacific).<sup>2</sup>

Ellis, in "Polynesian Researches," says: "Insanity prevailed to a slight degree, but individuals under its influence met with a very different kind of treatment. They were supposed to be inspired or possessed by some god whom the natives imagined had entered everyone suffering under mental aberration. On this account no control was exercised, but they were treated with the highest respect. They were, however, avoided," etc.

A distinguished writer and a graduate in medicine of this University told me that when he was in the South Sea Islands he bore a commission from a friend and former teacher to procure, if he could, a specimen of a Polynesian skull. He experienced much difficulty in the quest. At last he met a lonely man whom everyone seemed to avoid, and engaged his services for a small consideration. The man was true to his promise, and at the appointed time procured a suspiciously fresh-looking skull, refusing all information as to where he had obtained it. My informant was not a little uneasy over the transaction, but he was afterwards assured by a competent authority that the procurer of the skull was a lunatic, whose actions, however outrageous, none of his fellow-islanders would dare to question. Emin Pasha in

<sup>1</sup> Vol. i. page 205.

<sup>2</sup> Cook's Voyages, Vol. iii. p. 131.

his book, "Central Africa," says: "Insanity and also temporary mental aberration are frequent. The latter is treated with herbal remedies which effect an immediate cure by means of sleep and sweating."<sup>1</sup> Wilson and Falkin, "Uganda and Egyptian Soudan," state: "Temporary madness is pretty common, and generally lasts for three or four days, but persons thus afflicted do not become very violent."<sup>2</sup> I might go on indefinitely multiplying extracts from the writings of travellers to the same effect, but it would serve no additional purpose. When speaking of epilepsy and of hysteria in a previous lecture, I pointed out that no nation or race in the world was free from these affections, and that if there was any difference between races it was in the extent to which they were invaded by these diseases. If such diseases as hysteria cannot exist independently of the neuropathic constitution, and if insanity is equally dependent upon that constitution, then it is the constitution itself, and not any particular manifestation of it, which we ought to endeavour to trace. To my mind it seems conclusive that if a race is largely affected by hysteria, the individuals composing it must have a corresponding share of the neuropathic constitution in their heredity. I also believe that the lower animals are equally subject to pathological mental aberrations. One cannot read such articles as those written by the late Dr Lauder Lindsay of Perth, in the *Journal of Mental Science*,<sup>3</sup> or Pierquin's remarkable book,<sup>4</sup> without being convinced not only of the great variation of mental manifestations by animals of the same species, but also of the existence of pathological mental conditions among them. Further, a perusal of the mental condition induced in animals by removal of the parathyroid glands, as performed in the experiments of Herzen and Schiff, reveal the possibility of acute insanity in several species of animals.<sup>5</sup> But the important reason why statistics differ as between savage and civilised peoples is that the type of the race, the cerebral organisation, and consequently the mode of reacting to the environment, are all different. No doubt the same forms of mental disease are to be found among savages as among ordinary

<sup>1</sup> "Central Africa," p. 94.

<sup>2</sup> "Uganda and Egyptian Soudan."

<sup>3</sup> *Journal of Mental Science*, Vols. xii., xvii. and xxiii.

<sup>4</sup> "De la folie des Animaux." Paris, 1839.

<sup>5</sup> "Jeandelize l'Insuffisance Thyroïdienne." Paris, 1903.

civilised Europeans, with certain exceptions. A little consideration will show that insanity of such types must be more or less concealed by the want of culture and education among savages. For example, what can it matter to a savage tribe though one of its members should be the subject of hallucinations? Of what great interest would it be that he should profess to be possessed by the devil or bewitched? I might go a step further and ask, what would have been the result in Scotland three hundred years ago if an individual were to express delusions of persecution referred to unseen or occult agencies? Such a person would undoubtedly run the risk of being accused of communing with the devil, and might be burned alive. At the least he would be liable to the gentle persuasion of the thumb-screws and the boot. It is well known that in the days of Louis Philippe, no later than the early part of last century, when the power of the Church was supreme in France, it was considered prudent to conceal certain obscure or unusual symptoms lest a charge of witchcraft should be based upon them. What medical significance could the symptoms of melancholia have in a non-ethical and non-scientific society? In a previous lecture I pointed out that so far from the neuropathic constitution being considered pathological among barbarous people, its manifestations are regarded as valuable qualities, and are regularly put to a marketable use. The subjects of epilepsy and hysteria supply the ranks of the soothsayers, the magicians, the workers of miracles, and the priests. The contention, therefore, that neuropathy, which is the basis of the neuroses and of insanity, is less common among savage and barbarous people is wholly unfounded. There is, however, another reason why this belief in the immunity of the less civilised from insanity has obtained currency, and it is because no qualified person has ever been at the trouble to investigate the matter. About a year ago Professor Kraepelin, of Munich, went to Java and analysed the forms of insanity among the natives in one of the large asylums there.<sup>1</sup> His results, though not yet fully published, are highly significant in their bearing on this question. His first inquiry was whether the influence of climate and other tropical conditions of life modified in any way the symptoms of mental disease. He found, however, that Europeans born

<sup>1</sup> *Centralb. für Nervenheilk. und Psychiat.*, July 1904.

and reared in Java present exactly the same clinical types of mental diseases as at home. As to the abuse of stimulants and narcotics, the natives do not drink alcohol, and there were therefore no cases of alcoholic insanity among the native population of the asylum. Opium smoking and abuse of the drug is, however, common, yet no patients in the asylum owed their insanity to that cause. The same is true of the large asylum at Singapore, in which city the Chinese population is notoriously given to the abuse of the drug. Of especial interest also is the fact that, out of 370 insane natives, there was not a single case of general paralysis, whilst among 50 European men who were inmates of the asylum at the same time there were 8 cases. There is no satisfactory explanation of that fact on the basis of the ordinarily accepted theory of the causation of the disease, for the native of Java is not less the victim of syphilis than other similarly circumstanced people. For an explanation we must fall back upon such imperfectly understood questions as racial differences and race types. Dementia præcox was found to be extremely frequent, and on the whole presented similar symptoms as are found among Europeans. On the other hand, mania-melancholia was rare. Many cases seemed to bear a resemblance to it, but they were found on closer observation to be distinct and peculiar forms of epileptic or hysterical mania. In those cases in which there was no doubt in diagnosis, the symptoms presented several variations from the European type. Especially was this the case in the depressed form of the mania-melancholia syndrome, where many of the characteristic symptoms were wholly absent. For instance, ideas of "sinning" were never expressed, and maniacal agitation was less developed and more monotonous than is usual in Western Europe. The great difficulty experienced in forming a satisfactory diagnosis of mental affections in Java was the preponderating amount of "Amok" and "Latah" among the patients. The symptoms of these semi-hysterical diseases not only formed special clinical groups, but they appeared also to colour the character of other and distinct forms of insanity. As I mentioned in a previous lecture, latah is the great mental affection of the Malays, just as hysteria is the corresponding disease *par excellence* of the Samoyeds and Kamskatchens and other nations of North-Eastern Europe and

Northern Asia. Short, quickly passing hystero-maniacal attacks similar to those which Emin Pasha and Felkin describe among the natives of the Soudan, Kraepelin describes as frequent among the Malays. This leads to the conclusion that it is not so much a question of the frequency of insanity as of its type which ought to be the basis of enquiry when studying the manifestations of insanity among peoples widely separated in development, whether racial or social. We see then that statistical insanity in its fluctuations must depend upon the scientific standard of the people whose insanity is being considered, and upon their ethical attitude towards disease of all kinds. It cannot depend upon an absence of neuropathia or psychopathia, for the theory of variation forbids the possibility of the existence of a community in which the members are at a uniform level of physical or mental endowment.

I now come to the important, and I am sorry to say, the controversial question of the causes of insanity. It seems to me that I might conveniently dismiss this subject by adopting the laconic style of the student who was assigned the task of writing an essay on "Snakes in Ireland," by saying in three words, as he did, "There are none." But as the great majority of people still believe in the validity of the numerous causes which are popularly believed to create insanity somewhat in the same way as violent exercise causes fatigue, or eating salt fish causes thirst, it is necessary to refer briefly to the subject.

When we apply the word "cause" to insanity, we must mean one of two things: namely, either the cause of the whole hereditary and pathological processes which culminate in insanity; or the agencies which precipitate individual attacks of insanity in a psychopathic subject.

1. The causes which are supposed to produce insanity *de novo*. The fact that an individual may live free from any suspicion of mental unsoundness for the first two decades of life has undoubtedly tended to obscure our notions regarding the causation of mental disease, and has led us to seek for external explanations where a little consideration would enable us to perceive that in this respect insanity does not differ from other constitutional diseases such as cancer, phthisis, or gout. If a disease is transmissible from ascendants to descendants, its appearance must follow the course of the development of the individual,

otherwise we should meet with cancer and angina pectoris in childhood, while rickets and the disorders of dentition should be equally common in the later as in the earlier decade of life. Insanity not being one disease, as we have seen, but a heterogeneous group of many diseases, we cannot speak correctly of its "cause," any more than we can intelligibly speak of a cause of the diseases of children or a cause of tropical diseases. It is worse than useless, therefore, to publish tables of the causation of insanity which make no reference to the special affection which the cause is supposed to produce. I say "supposed," because causation can at best be only a matter of opinion. What do we mean when we ordinarily use the word "cause"? We must mean one of two things. Either that because two phenomena occur regularly in a certain sequence they therefore stand to each other in the relation of cause and effect. Or that because two phenomena occur in association, the one necessitates the appearance of the other. The fallacies of the first proposition are that, if it were universally true, we should be compelled to the conclusion that because day and night regularly succeeded one another, the one was the cause of the other. Obviously, day and night stand in no such relation to one another, but the same fact is not always so clear in the case of many other natural processes with which we are less perfectly conversant. Again, it is not possible to isolate any single phenomenon in sequence of events and call it a "Cause," for one thing depends upon another in infinite regression back into eternity. The possible fallacies in the second case are numerous. Among the simultaneously occurring phenomena we select one in which we are most interested, and call it the "Effect," and then we look for the "Cause" among the other simultaneously occurring phenomena. Such a quest is natural and in accordance with the constitution of the human mind. Yet though the process is the source of all human knowledge, it is no less the source of all error. If the human mind were perfectly co-adapted to natural phenomena there would be no danger of mistake, for knowledge would be perfect and speculation unnecessary. But as we are mentally constituted we almost invariably fall into error through selecting, not the true cause, but the one which we would rather believe to be the true cause. It is a well-known fact that when a person is hypnotised and told to do a certain action, say an hour after-

wards, he will perform the action without the least recollection that it has been suggested to him; but when he is asked why he did it, he will furnish a number of plausible reasons just as if he had spontaneously performed the act for sufficient conscious reasons. That is, I think, one of the most wonderful and suggestive facts in hypnotism. It explains our insatiable desire to find a cause for everything, and it also explains why we are generally mistaken. In order to be able approximately to assign a cause to any effect, it is first of all necessary thoroughly to know the nature of the effect itself. If we do not know what insanity is, how can we pretend to know what its causes are? It was the profound ignorance of the disease which led to the theory of its supernatural origin, and it was only in proportion as our knowledge increased that we have been able to formulate more rational ideas of its associated phenomena. But as our knowledge of the fundamental facts of insanity is as yet only fragmentary, our opinions regarding its causation are still necessarily crude and imperfect. All we can honestly claim to assert is that certain symptoms have been preceded or accompanied by certain definite phenomena which have been ascertained to precede or accompany similar symptoms in a fixed and constant ratio. Take, for instance, the use of the word cause as applied to the numerically expressed relation of syphilis to general paralysis. In the first place, we do not know enough about general paralysis to assign it to any one cause; and in the second place, syphilis cannot be the cause of general paralysis, for only an extremely minute fraction of the subjects of syphilis become general paralytics. Alcoholism was discussed in the third lecture, where it was, I think, successfully proved to be a neurosis closely allied in its symptomatology and heredity to the other neuroses and to insanity. That being so, its prominent place in a classification of insanity need be a matter of no surprise. Its combined homologous and dissimilar heredity is between 80 per cent. and 90 per cent. of the cases of alcoholic insanity. It may seem absurd to discuss the question of the ætiology of alcoholism, for, of course, if there were no alcohol there could be no alcoholism. Yet even here, so complicated are the causes of mental disorder, that it is necessary to point out that the true cause is a defective heredity which (1) induces the subject to crave for a particular mental state—not for alcohol,



but for the state which alcohol most conveniently produces ; (2) which provides the subject with a constitution which is particularly susceptible to the influence of such poisons as alcohol ; and (3) which is in many cases the cause of a mental unsoundness independent of alcohol.

2. The causes which precipitate individual attacks of insanity.

If we restrict the term "causes of insanity" to those agencies which precipitate individual attacks in predisposed persons, we are on more limited and less important grounds. There are, no doubt, innumerable such agencies, but I question if a tabulation of them is so profitable or so beneficial as some people are apt to believe. Take, for example, the causes of an epileptic fit. In the same individual different agencies at different times determine the onset of a seizure. At one time it may be alcohol, at another time an error in diet, at another time a violent mental impression, while at other times the fit comes on without any assignable cause—merely as a manifestation of the morbid periodicity of the disease. Is it not a misuse of language to call these and similar agencies "causes" of epilepsy? It is the same with insanity. "When we are told" (says Maudsley) "that a man has become mentally deranged from sorrow, need, sickness, or any other adversity, we have not learned much if we are content to stay there; how is it that another man who undergoes an exactly similar adversity does not go mad?" The great question of the present day and one which is constantly asked by the public is, "What are the causes of the increase of insanity in the population?" The obvious reply is to ask in return whether there is any real increase of insanity. I have fully convinced myself (I do not know if I have succeeded in helping you to the same conclusion) that there is no increase of insanity. I am willing, however, for the sake of argument, to look at the question in another form, and ask why is not the proportion of insanity less than it is? The causes generally assigned are those which produce physical deterioration of the members of a community. These are overcrowding, density of population, improper food, alcohol and physical diseases. It might be pertinent, before discussing this question, to ask whether physical deterioration, in the sense in which the word is popularly understood, really occurs at all. Very grave doubts have been

expressed on the subject by competent authorities, and no proof of such deterioration has been brought forward. When it is seriously argued, however, that alcohol, improper food and diseases of all kinds do not so much produce insanity in the *first* generation, but by their action on the sexual elements of the parents induce an unstable condition in the brains of the next generation,<sup>1</sup> in short, that these evil influences have caused variation in an unfavourable direction, all that can be answered is that such statements are founded on speculation, and should not therefore be put forward as a working scientific hypothesis. I do not profess to know the causes of variation, but it is evident that the promoters of this theory have misunderstood the meaning of the term variation. The word, as scientifically understood, means divergence from a standard mean of any quality. It implies divergence to both sides of the mean; therefore it is both good and bad. If the mean height of a population is 65 inches, then for every man that is 60 inches in height there is another 70 inches high. On this hypothesis one is equally justified in asserting that alcohol causes genius as that it is a cause of genetic insanity. Even if we assumed that alcohol directly increased the amount of genetic insanity in a community, we would have to satisfy ourselves also as to whether the drinking habits of the people in that community were increasing or decreasing. If the historical accounts of the drinking habits in this country are not fallacious and garbled, there can be no doubt that the people are much more sober than they were 150 years ago, and are steadily becoming more sober year by year.<sup>2</sup>

On the whole, while we may be ready to acquiesce in deploring the insanitary, diseased and alcoholic condition of many portions of our urban population, the question in the light of statistics does not bear upon the increase of insanity in any way that I am able to make out. On the contrary, there is evidence which goes to show that, whatever the social and moral condition of a people, a strong and effectual effort is made by nature to counteract these inimical surroundings.

I select the following striking remarks from the evidence

<sup>1</sup> Dr Wigglesworth, "Evidence Phys. Det. Com." (8988); and Dr Ford Robertson, *Brit. Journ. of Inebriety*, 1904, p. 104.

<sup>2</sup> *Vide* Samuelson's "Hist. of Drink"; and Shadwell, "Phys. Det. Committee" (12280-86).

given before the recent (1904) Commission on Physical Deterioration.

Dr Eichholz said :—(566) “The number of children born healthy is, even in the worst districts, very great. The exact number has never been the subject of investigation, owing largely to the certainty which exists upon the point in the minds of medical men ; but it would seem to be not less than 90 per cent.” (646) “The percentage of badly born children among the poor is not sensibly greater than among the rich, and such diseases as are hereditary, such as insanity and neuroses, in which we include alcoholism and other inherited diseases of bad living, affect the one as much as the other.” Professor Malins of Birmingham, President of the Obstetrical Society of London, said :—(3124) “I think the testimony of experienced observers would be in accordance with the views expressed by Dr Eichholz, though perhaps not to such a large extent. I should say that from 80 to 85 per cent. of children are born physically healthy.”

If the poorest and most ill-nurtured women bring forth as hale and strong-looking babies as those in the very best conditions, the interpretation would seem to follow that Nature gives every generation a fresh start. It must also follow that environment has very little to do with the ante-natal condition. Children, it would seem, are not necessarily born degenerate, though born in the most sordid surroundings, and though born of parents who have acquired evil habits of life or unsound bodily health. All that the majority of these children require to make them average citizens is a chance in life—a chance to escape into better, healthier and cleaner moral and social surroundings. I am confirmed in this statement by the marvellous results obtained in Glasgow by boarding out “slum” children in the houses of the peasantry throughout Scotland, regarding which we heard a good deal at the International Congress for Home Relief held in this City last June. We were informed on good authority that about 80 per cent. of these children turned out well in life, which is saying more than even an optimist would be inclined to say of ordinary children.

**THE PATHOLOGY OF FRIEDREICH'S ATAXIA.**

By HARRY RAINY, M.D., F.R.C.P.E.

THROUGH the kindness of Dr Mackie Whyte of Dundee, I have received the post-mortem specimens of two of the cases of Friedreich's Ataxia which he described clinically in 1898,<sup>1</sup> and as the examination of one of these has emphasised certain facts of considerable interest in the pathology of the disease, it seems desirable to bring forward these facts without delaying until a complete investigation of all the material has been accomplished. A full criticism and review of the literature of the subject will be reserved for a second paper; meanwhile, however, a few of the leading observations may be referred to, before passing to the consideration of the specimens which have been personally examined by myself.

**I. HISTORICAL.**

As early as 1861, Friedreich had described the characteristic change in the posterior columns, and in the succeeding years the alterations which occur in the lateral, and at times in the anterior, columns were successively indicated. By the year 1877 a considerable number of papers had appeared on this form of ataxia, and these were reviewed by Rütimeyer, whose analysis of the work of other observers, supplemented by his own researches, added considerably to the knowledge of the typical lesions. He emphasised the fact that the condition was one which involved both the nerve structures and also the interstitial tissue within the cord, and he further described changes in the posterior nerve roots and in Clarke's column. During the following years, as the number of undoubted cases of the disease grew more numerous, further contributions were made at frequent intervals both to the clinical history and the morbid anatomy of the condition, and in 1890 these were collected by Ladame in a critical digest which supplied full details of all important cases up to 1889. In the same year Déjerine and Letulle undertook a more careful histological study of the nature of the sclerosis than had been possible with the less specialised methods of earlier days, and concluded that the sclerosis of the posterior

<sup>1</sup> Whyte, J. Mackie, *Brain*, vol. xxi. p. 72, 1898.

columns was essentially neuroglial in origin, and was characterised by the presence of whorls or "tourbillons" which were analogous to those which had previously been described as occurring in the brains of certain epileptics. They believed that the sclerosis of the lateral columns was of a different kind, arising essentially in the connective tissue which penetrates the cord from the pia mater, whilst the vessels which entered the cord with it also showed characteristic changes.

At the time many observers failed to confirm their opinion as to the peculiar character of the sclerosis in the posterior columns, but gradually corroborating evidence has accumulated. In 1893, Senator suggested that the disease might really originate in cerebellar defect, and this led to a more careful examination of the cerebellum. The results in certain cases supported Senator's views, but instances were also put on record where the cerebellum appeared to be perfectly normal. About the same time (1893), Pierre Marie isolated a special type of hereditary ataxia in which, owing to the characteristic inco-ordination that was observed, he was led to the conclusion that the lesion was essentially cerebellar. The relation of this Hereditary Cerebellar Ataxia to the typical form of Friedreich's disease is still under discussion; some authorities emphasising their points of difference, others laying more stress on their resemblances.

In 1896, Tedeschi summarised the literature that had appeared up to the previous year, and his careful and critical paper served as a starting-point for further researches.

In 1897, G. Bonnus described the post-mortem findings of a case which had presented very typical features during life. He found that the cerebrum, the cerebellum, the upper part of the medulla, and the meninges of the cord, were quite unaffected. The usual changes were present in the spinal cord, and, as regards the motor tract, passed up for some distance in the lower medulla. The posterior nerve roots were implicated, and the peripheral nerves showed corresponding alterations. Some of the arteries entering the cord itself were observed to be affected.

In the same year Adolf Meyer described the changes found in a case that had been clinically observed by Dr Sanger Brown. Amongst other points he called attention to the presence of numerous "corpora amylacea" in the affected portions of the

cord, and showed that they were particularly abundant near the entry of the posterior nerve roots. He also confirmed the fact that much of the sclerosis was of neuroglial origin.

In 1898, H. Mackay recorded a case in which he found degenerative changes in the spinal ganglia, and confirmed the observation that the morbid process in the posterior columns had spared the endogenous tracts much more than those directly related to the entering posterior roots.

In this year also Léon Bonnus reported fully a case of long standing, in which the cerebellum appeared perfectly normal both microscopically and to the naked eye. He noted very distinct changes in the peripheral nerves, which were particularly evident in those that contained a large proportion of sensory fibres.

In 1899, G. E. Rennie reported the case of a child aged twelve years who died of an intercurrent affection when symptoms of ataxia had only existed for fifteen months. The lesions were sharp and definite, corresponding closely with those found in cases of relatively long standing. This observation seems important as showing how early the characteristic lesions develop, and suggests that they are definitely associated from the beginning.

In 1901, Philippe and Oberthür, using modern histological methods, examined carefully into the nature of the sclerotic change, and were able to confirm the view that it was essentially neuroglial, although they saw ground to believe that the connective tissue and vessels participated also.

In 1903, a very elaborate and detailed examination of two of Dr Sanger Brown's cases was made by Dr L. F. Barker. The report contains a full description of all parts of the nervous system, and gives accurate measurements where these are desirable. In the main it corroborates Meyer's work, but additional facts were observed, amongst which the most important is that marked degeneration was found in both the grey and the white substance of the cerebellum. A still more recent paper is that of Professor Mingazzini and Dr Perusini, the first instalment of which was printed in the latter part of 1904. They failed to detect any change in the cerebellum, but describe the pia mater of the cord as being distinctly thickened over the affected regions.

Summing up the facts recorded above, and arranging them

in a more systematic order, we may epitomise the present state of knowledge as follows:—

1. *Cerebrum*.—No changes have been described which are of any material importance.

2. *Cerebellum*.—In many cases it has been described as normal. In other cases degenerative changes or defects in development have been recorded. In a certain number of instances there is insufficient evidence of thoroughness in the examination, but most of those of recent date which are reported as normal have been examined by thoroughly competent observers.

3. *Medulla*.—Changes have been recorded in a fair number of cases. These mainly occurred in the motor tracts, and were most frequent in the lower portion of the medulla.

4. *Cord*.—The posterior columns, especially the columns of Goll, are characteristically involved, the endogenous tracts being much less affected than those containing fibres derived from the posterior nerve roots.

The lateral columns show involvement of the direct cerebellar tract, the crossed pyramidal tract, and part at least of Gowers' tract. Evidence of considerable weight has been adduced to prove that the *motor* fibres are really implicated in the sclerosis of the crossed pyramidal tract.

The direct pyramidal tract has been found affected in a relatively small number of cases.

The cells of Clarke's column are invariably degenerated, and the posterior cornua are often shrunken.

Little or no change has been seen in the motor cells of the anterior cornua, though in a few advanced cases slight alterations may have been found.

The cord as a whole has often been described as smaller than usual.

The nerve elements which have been involved are replaced by a tissue which is mainly of neuroglial origin, and which contains in many instances "whorls" and "corpora amylacea." Along with the neuroglial proliferation there is often an increase of connective tissue and alteration of the blood-vessels which enter the cord with it.

In certain cases the pia mater has been found to be thickened, in others it retains its normal proportions.

The posterior nerve roots are involved in the sclerosis to a very marked degree, and the cells of the posterior root ganglia have been observed to be degenerated.

The anterior nerve roots appear to escape entirely.

5. *Peripheral Nerves*.—In several cases these have been found degenerated, and the degeneration has been proportional to the ratio of sensory fibres they contain.

## II. REPORT OF CASE.

The case from which the specimens now reported upon were obtained, was described as No. II. (H. M'A.) in Dr Mackie Whyte's paper. It was of fairly long standing, symptoms having been observed for about twenty-two years before the fatal termination. The histological examination was conducted in the Laboratory of the Royal College of Physicians, Edinburgh, and I have to express my indebtedness to Dr Noël Paton for the facilities which were there placed at my disposal.

The post-mortem examination was made by Dr Mackie Whyte, who removed the brain, cerebellum, spinal cord, peripheral nerves, and parts of various muscles. After fixation in Müller's fluid they were forwarded to the laboratory in Edinburgh. Unfortunately some posterior root ganglia which had been isolated were lost in transit, so that no examination of them could be made.

The specimens were cut in celloidin and stained in various ways: the nerve tracts chiefly by Weigert Pal's method, the neuroglia, vessels, connective tissue, and degenerated structures by numerous other processes specially adapted to the ends in view. At all important regions the sections were cut and mounted serially; in less important situations every tenth section was mounted and the rest reserved for contingencies which might arise in the course of the research.

The nerve cells were stained in some specimens, but owing to the fixation in Müller's fluid, considerable reserve had to be exercised in drawing conclusions regarding their finer structure.

The systematic examination of the various specimens yielded the following results:—

1. *Cerebrum*.—On naked eye examination the surface appeared normal. The convolutions were well developed, and



there was no appearance of atrophy or other change. The interior parts of the cerebrum also presented a healthy aspect. Microscopical sections of the motor cortex showed that the pyramidal cells and other elements were present, and, so far as the methods of preservation of the specimen allowed one to form an opinion, were perfectly normal. Weigert Pal preparations of the various bundles of motor and other fibres passing through the internal capsules and below that to the crura cerebri stained clearly and revealed no abnormality.

2. *Cerebellum*.—On naked eye examination the cerebellum appeared to be normal in size and in the proportion of its several parts. No microscopical changes were detected either in the central or in the lateral lobes. A specially careful examination was made of the lower cerebellar peduncles, but nothing unusual was detected, and there was no indication of any sclerosis, such as appears in the direct cerebellar tract in the cord.

3. *Pons and Medulla*.—These appeared unaltered on naked eye examination. On microscopical examination the motor tract remained intact until it had reached the level of the decussation of the pyramids, at which level the sclerotic change became visible.

4. *Spinal Cord and Peripheral Nerves*.—*Naked eye examination*.—The nerves and muscles did not appear in any respect abnormal. The spinal cord was slightly smaller than one usually finds it in health. In the dorsal region it was much distorted, but part of the distortion may have been due to the difficulty in removing it at the autopsy, as the spinal column was greatly and irregularly curved at this level. *Microscopical examination*.—Sections of the muscles disclosed no abnormality, and so far as could be determined, they were unaltered in structure by the disease. The peripheral nerves showed partial degenerative changes; some bundles of fibrils being much implicated, whilst others were unaffected. Probably the latter were those whose function was a motor one, whilst the former were sensory. The state of the posterior root ganglia could not be determined, as unfortunately they were not included in the material available for examination. The nerve roots themselves were very characteristically affected, the anterior roots appearing perfectly normal, whilst the posterior ones exhibited very



FIG. 1.

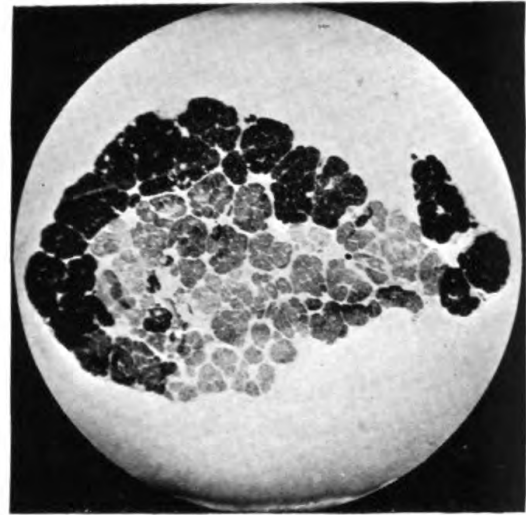


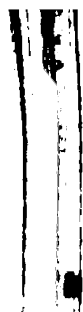
FIG. 2.



FIG. 3.



FIG. 4.



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advanced degeneration. The degeneration extended over all the posterior roots, from and including those of the cauda equina up to the upper dorsal region. Above this the process was slightly less uniform, some of the roots containing a considerable proportion of healthy fibres, whilst others were almost wholly degenerated.

In the cord itself the *posterior columns* were found to be affected from the sacral segments upwards. The diseased areas corresponded to the continuation, in the cord, of the posterior nerve roots which had been found degenerated; whilst the endogenous tracts escaped, except when they were involved by secondary changes, induced by their proximity to affected parts. In the upper dorsal region, where the nerve roots were less uniformly implicated, healthy root fibres were seen to enter the cord, and were visible in the higher sections which were obtained in the cervical region. The lower cervical region contained some very degenerate nerve roots, and their prolongation in the cord was filled in by interstitial tissue. This tissue could be seen in the cord sections as a "boomerang-shaped" area, which corresponded to the position normally occupied by the fibres ascending from these roots. Some of the highest cervical nerve roots were healthy, and their fibrils could be traced upwards to the nuclei at the top of the column. Above these nuclei the morbid process did not extend to the sensory tracts passing upwards in the medulla. In the *lateral columns* there was marked degeneration of the crossed pyramidal tracts, of the direct cerebellar tract, and, in part, of Gowers' tract. The motor fibres, as has already been noted, seemed quite normal above the level of the decussation of the pyramids, though possibly the number of fibres may have been fewer than usual. This definite limitation of the degeneration, which has been observed in other cases, is not very easy of explanation, though it is not impossible that it may be associated with the transition from the cerebral to the spinal type of vascularisation.

It has been suggested by some authorities that the motor fibres in the crossed pyramidal tract are not truly involved, but that the lesion really arises in fibres of different functions which lie amongst the motor fibres of this tract. In the present instance the degenerative changes were very advanced and certainly implicated all the nerve elements in the tract, and the

sclerotic change appeared to correspond closely in nature to that which involved the posterior columns, although the process was hardly so advanced as in the latter situation.

Jules Vincelet, working under Marie's guidance, has recently drawn attention to the early appearance of the "extension type" of Babinski's sign as an indication that there is involvement of the true crossed pyramidal fibres, and not merely of other fibres which are associated with them but exercise other functions.

The *anterior columns* in the case under consideration were unaffected, the direct pyramidal tract remaining intact.

The interstitial tissue which replaced the nerve elements in the affected regions was shown to be of neuroglial origin by its reaction to the special stains, such as van Gieson's, which differentiate between neuroglia and ordinary connective tissue. In certain parts, where the process had advanced to a certain degree, but where it had not reached its full development, the whorled arrangement described by Déjerine was frequently met with. The most abundant instances were found in the posterior columns, but a sufficient number of examples were observed in the region of the crossed pyramidal tract to convince one that the sclerotic process was essentially identical in both situations.

Where the change was furthest advanced the whorled appearance could no longer be traced, but was replaced by a dense felted growth of interstitial tissue, which tended to pass beyond the original limits of degeneration, and to implicate and strangle healthy nerve fibrils in unaffected parts.

The vessels and pial septa of the cord were not obviously thickened or otherwise altered in the diseased areas; but the systematic measurements, which alone are adequate to settle the question definitely, have not yet been completed, and a definite report on these will be supplied in another communication.

"Amyloid bodies" which, following Dr Ford Robertson's opinion, I am inclined to regard as mesoglia cells more or less degenerated, were extremely abundant in the affected areas, and particularly so in the posterior columns. This last situation is, of course, the region where they are most numerous in healthy cords, but in this disease they appear to be vastly more numerous than they ever are in health, as can readily be seen from the specimen figured amongst the illustrations attached to this paper. In Müller-hardened specimens they can be well demonstrated by



FIG. 5.

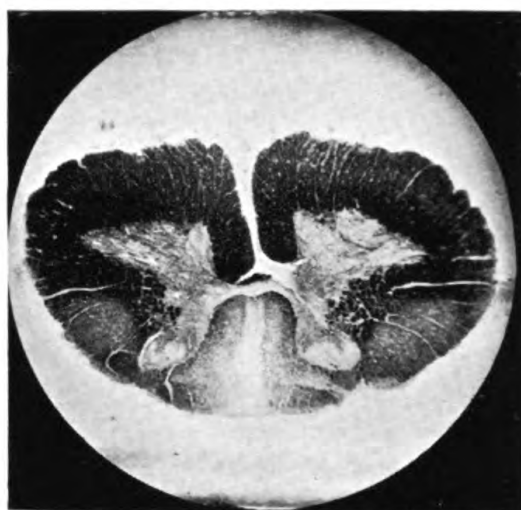


FIG. 6.

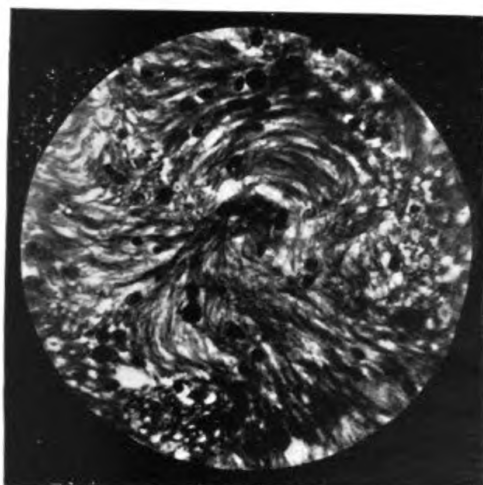


FIG. 7.

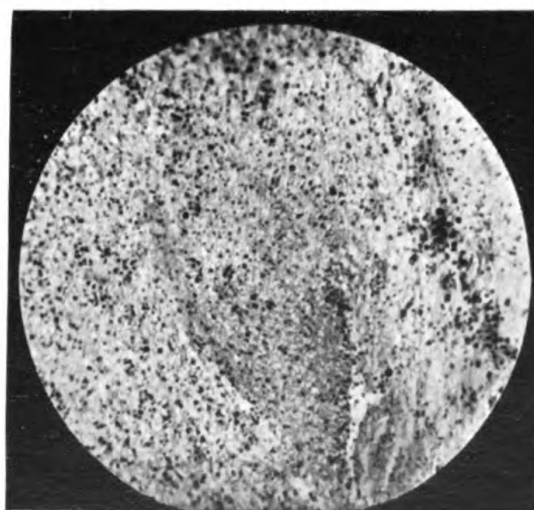


FIG. 8.



an Orcein stain. They vary greatly in size, and the explanation of this may be found in the shape of the degenerated mesoglia cells, of which they are cross-sections.

In the grey matter of the cord the following are the main facts which I observed. The cells of Clarke's column were in a state of advanced degeneration; in many sections taken from the levels where they are most abundant in the healthy cord hardly any could be found, and none of those that were seen looked healthy. The other nerve cells of the posterior cornua also seemed poorly developed, but they have not yet been fully investigated. No material deviation from the normal was observed in the motor cells of the anterior cornua, but the pericellular nerve reticulum seemed rather less abundant than usual—a state of matters not unlikely to occur when the degeneration of the crossed pyramidal tracts was so far advanced.

Owing to the fact that the specimens were all hardened in Müller's fluid, the exact degree of cytological change could not be very accurately determined, but the grosser structural alterations were identified with certainty.

The most important fact which the above observations contain is that towards the upper limit of the lesion in the posterior columns some of the posterior roots were healthy and others degenerated, and that the fibres which are continued upwards in the substance of the cord from these roots were found to correspond, as regards their state of health or implication, with that of the roots from which they arose. Thus, as will be seen from the sections in the lower cervical region of the cord which are figured at the end of this paper, to the degenerated roots of the seventh cervical nerves corresponds an area of ascending degeneration bordered on either side by relatively healthy nerve fibres. This "boomerang-shaped" area could be traced for some distance upwards in the cervical cord in the region corresponding to that which has been assigned to the ascending fibres emanating from the seventh cervical root.

The conclusion which one may legitimately draw from this observation is that the lesion is primarily one of nerve tissue and not a disease of the neuroglia, and that certain continuous elements of the nerve tissue are selectively implicated. The fact that the lesion affects not only the posterior nerve roots and their upward



continuation in the cord, but also certain fibres of the peripheral nerves, confirms this opinion.

It may be worth while directing attention to the fact that questions bearing on the distribution of a lesion like that found in Friedreich's ataxia can only be satisfactorily determined by the somewhat tedious process of preparing long groups of serial sections from the whole length of the cord in which the disease is found, and that the most important region to study in detail is that which marks the upper limit of the lesion, as here one is most likely to encounter these transitional stages which are best fitted to throw light on the exact nature and method of invasion of the pathological process.

One may, for the present, sum up the opinions which the examination of this case seems to justify under five heads:—

1. A typical case of Friedreich's ataxia of long standing (twenty-two years) may show no changes in the brain or cerebellum, therefore changes in these parts of the nervous system do not constitute a *necessary* part of the pathology of this disease.

2. The appearances of the diseased tracts in the posterior and lateral columns of the cord are sufficiently similar to incline one to the belief that in both they are consequences of the same pathological cause.

3. The distribution of the lesion, especially as it is found in the posterior column and sensory nerves, indicates that the nervous elements are themselves primarily involved, and that the increase in interstitial tissue is secondary.

4. The cause of the disappearance of the nerve fibrils is to some extent a matter of speculation. So far as the writer's observations have hitherto gone, the evidence is distinctly against its resulting from changes in the pia mater or vessels of the cord, and one is left to assume that certain of the nerve elements are developmentally defective. This defect may be due to imperfect myelin production, or to a condition that for want of more definite knowledge may be called deficient longevity, or to an imperfect faculty of repair under the strain of continued use. It is also conceivable that some of the nerve elements may be specially susceptible to the action of a hypothetical toxin which might be postulated as being formed in this disease, but of such a toxin we have absolutely no evidence.

Of course the ultimate problem that confronts one is why

certain nerve tracts should show a special lack of vitality in a hereditary disease like Friedreich's ataxia, but though this is a problem of the utmost importance, the clue to its solution is still unattained.

5. The degenerated nerve structures in the cord are replaced by neuroglial elements; the earliest stage consists in the appearance of fibrils of interstitial tissue. These, as the process advances and cicatrisation occurs, form whorls or "tourbillons" which catch up and distort or destroy healthy nerve fibres. Finally, where the sclerosis is most advanced, the whorls coalesce into a felted mass of fibrils that occupy the whole substance of the most fully degenerated areas.

#### EXPLANATION OF PLATES.

- FIG. 1.—Peripheral nerve, stained by Weigert-Pal method. Some bundles of fibrils appear to be degenerated, and fail to stain well. They probably consist chiefly of sensory fibrils.
- FIG. 2.—Cauda equina, stained by Weigert-Pal method. Motor fibres healthy. Sensory fibres degenerated.
- FIG. 3.—Spinal cord, below fourth lumbar segment. Motor nerve roots healthy, sensory degenerated. Endogenous tracts of posterior columns escape.
- FIG. 4.—Spinal cord, about second dorsal segment. Sensory nerve root at this level fairly healthy.
- FIG. 5.—Spinal cord, at seventh cervical segment. A degenerated sensory nerve root entering.
- FIG. 6.—Spinal cord, at sixth cervical segment. The degenerated root entering in the seventh segment is here represented by a "boomerang-shaped" tract occupied by its degenerated ascending fibres.
- FIG. 7.—A "tourbillon" or whorl of neuroglial tissue in a degenerated area in the posterior columns.
- FIG. 8.—"Amyloid bodies" in a degenerated part of the posterior columns. Orcein stain.
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**FAMILY SPASTIC PARALYSIS ASSOCIATED WITH  
AMYOTROPHY.**

By GORDON HOLMES, M.D.,

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and Epileptic, London.

THE following two cases of an extremely rare condition seem to be worthy of record. The elder girl was an in-patient in the National Hospital under the care of Dr Ferrier, to whom I express my thanks for permission to publish the case, while her sister was at my request brought up from the country for examination and was consequently only seen on the one occasion.

The parents of the family are both alive and healthy and no history of any form of nervous disease could be obtained in the direct line. A daughter of the father's sister, however, is said to have been affected in much the same manner as my elder patient; she did not learn to walk till her sixth year, and when older got about only with difficulty. Her legs were stiff and tended to cross, and her hands are described as having been weak and useless, but I could not ascertain that there was any wasting of their muscles. The affection progressed till she died at seventeen years of age. As there was considerable mental deterioration, and as the girl was subject to epileptiform fits, the condition was more probably a cerebral diplegia than the disease which affects my patients.

There are five children in the family, all of whom are alive and well: D. S., aged 15 years, the elder patient; E. S., aged 13 years, the second patient; and three sons aged respectively 10, 7 and 5 years, who are said to be strong and healthy and to walk and run as other children. The mother has not had any miscarriages, and no evidence of syphilitic infection was obtainable.

CASE I. D. S., aged 15 years, is the eldest child of the family. Her birth was natural and as an infant she seemed strong and healthy. In childhood she had measles and pertussis, but neither seemed to leave any after-effects. Nothing abnormal was noticed till she began to walk when two years old. Then it was observed that she was unsteady on her feet and frequently fell, being especially liable to trip up over the slightest obstacles

She could not play games as other children and has never been able to run. The difficulty in walking has increased from her eighth year, more rapidly during the past three or four years. At present she cannot walk more than a mile, tires easily and when tired complains of stiffness and aching of her legs. Some wasting or smallness of the muscles of the hands was first observed when she was about five years old, and has become more noticeable during the past four or five years. She has for some time now been unable to write and can sew "only after a fashion." Recently she has been liable to allow what she has carried to fall, and when her hands are cold they become quite useless. Her speech has always been unnatural, and for the past few years her mother has noticed "dancing of her eyes" when she gazes intently. There has been occasionally incontinence when she has had to retain her urine for long, but no other sphincter trouble.

She is described as bright and intelligent, but was rather backward at school owing to her difficulty in writing.

During the year she has been under observation the weakness of both upper and lower extremities has increased.

*State on Examination.*—A healthy-looking and well-nourished girl. Visceral organs normal. Intelligence and general psychical state fair for one of her social standing and education. No evidence of congenital syphilis or other constitutional disease. Special senses normal and the optic fundi are quite natural. No paresis of the ocular movements, but there is irregular nystagmus on looking to the right. Pupils equal and react well to light and on accommodation. Expression is rather fixed and vacant, with some rigidity and general weakness of both expressional and volitional facial movement. The tongue is also spastic and is moved about more slowly and awkwardly than is natural. Articulation is monotonous, toneless and nasal, though the palate is not paretic.

The musculature of the neck and trunk is fairly developed; she moves both head and body easily in all directions. The muscles of the shoulder-girdles and arms are also well developed, though not large, and their strength is relatively good. The forearms are smaller and there is some falling away along their ulnar borders, while practically all the small muscles of the hands are very much wasted, and owing to the disappearance

of the thenar and hypothenar eminences the palms are flattened. The hands are constantly held in the typical "claw-position," the fingers being extended at their proximal and flexed at their distal joints, so that they cover the thumbs, which lie in the palms. The flexors of the wrists are strong, but the extensors so weak that on firmly grasping anything the hand flexes. The strength of the long flexors is excellent, but the long extensors cannot straighten the fingers to the normal extent. The interossei, which are considerably wasted, cannot extend the fingers fully at the interphalangeal joints, and adduction and abduction of the fingers is weak and limited. The abductor indicis and abductor minimi digiti are also weak, they can only just move their respective fingers. The three long extensors of the thumb act well, but this is only flexed by the long flexors and the movements of abduction and opposition are almost lost, though adduction is fair. It would seem that the weakness of the various movements is in part due to a slight degree of rigidity which is demonstrable at the shoulder and elbow-joints.

The muscles of the buttocks and thighs are of fair bulk; there is perhaps some thinning above the knees. Both the calves and the anterior tibial muscles are uniformly wasted and feel abnormally firm and tough to touch. Each foot is in the position of pes cavus and talipes equinus and cannot be dorsiflexed beyond the right angle owing to contracture of the tendo Achilles. The toes are over-extended at the metatarsophalangeal joints and flexed at the interphalangeal. There is considerable rigidity and stiffness of both limbs, but it is easily overcome by passive movement. All movements are weak in relation to the bulk of the muscles, more especially dorsiflexion of the feet. There is no fibrillation in the muscles of either the upper or lower extremities.

*Gait.*—She walks bent forwards with the arms abducted from her sides and moved about so as to balance her. The feet are raised rather too high, but frequently dragged along the floor. The gait is ungainly and unsteady, but this is due to the weakness and rigidity of her legs rather than to ataxia. The feet are widely separated and she can scarcely stand when they are approximated. The heels come easily to the ground.

The faradic and galvanic excitability of the wasted muscles

of the hands is much reduced, lost in some of the intrinsic muscles of the thumbs, but elsewhere the electrical reactions are normal.

No change of any form of cutaneous excitability could be detected and the sense of position is intact in all limbs. There was no visible trophic changes except that the skin is rough and ichthyotic and the feet constantly cold and cyanosed. All tendon-reflexes are exaggerated and patellar clonus could be obtained in each leg; but ankle-clonus, probably owing to the contracture of the tendones Achilles, could not be elicited. The abdominal reflexes are absent and plantar stimulation gives definite extensor responses.

CASE II. E. S., aged 13 years, was born naturally. She had measles and pertussis in infancy, but has otherwise enjoyed excellent health. She commenced to walk when fourteen months old, but never got about naturally or as other children, and has never been able to run. Was always very liable to fall or trip if she hurried.

The weakness of her legs increased for some years, but has recently been more or less stationary. She can only walk short distances, tires easily, and when tired drags her feet along the ground. Nothing abnormal was noticed in her hands till it was found out that she could not learn to write properly and that they were extremely awkward in finer actions. The affection of the upper extremities has not increased recently. There has never been sphincter trouble and no peculiarity of voice or facial expression has been noticed. Her mother describes her as a bright and intelligent child, she has been easily able to keep up with her class-mates at school.

*State on Examination.*—A well-developed and healthy-looking girl, visceral organs normal and intelligence fair.

Her special senses and the functions of the cranial nerves are normal. Ophthalmoscopic examination reveals nothing unnatural in the eyes. There is no nystagmus. Her facial expression is rather fixed and vacant, but her smile is more natural than her sister's. There is no definite peculiarity in articulation.

The muscles of the neck and trunk and those of the shoulder-girdles and arms are well developed and uniformly large, but the forearms are relatively smaller, and the extensors of the wrists,

especially of the right, are definitely weak, though the hands do not flex when she grasps firmly. The muscles of the thenar and hypothenar eminences are much wasted, the atrophy being greater in the right hand than in the left. The hands assume the same posture as her sisters, but the mal-position is neither so great or so constant. The thumb is also rotated outwards so that its flexor surface is in the plane of that of the fingers. The long flexors and extensors of the fingers are strong, but the interossei are very weak; she cannot straighten the fingers at the interphalangeal joints and they tend to be over-extended at the metacarpophalangeal. Adduction of the thumb is fairly easy, but abduction, opposition and flexion are very feeble.

All movements of the upper extremities are well co-ordinated and no tremor or spontaneous movements have been observed nor is there any appreciable rigidity of the limbs.

The muscles of the hips and thighs are fairly large, those of the legs and feet relatively smaller, but there is no definite local wasting. The feet are in the position of pes cavus with some tendency to talipes equinus, but there is not much contracture of the calf muscles, and the heels come to the ground as she stands. Both limbs are slightly rigid and the strength of their movements is poor in relation to the bulk of the muscles. The co-ordination of movements is unimpaired. Her *gait* resembles that of her sister, but she walks considerably better. Her basis is broadened by the separation of her feet. She is liable to trip or stumble over any obstacle and occasionally drags her feet. The spastic affection of her legs is more evident as she ascends or descends stairs.

The electrical reactions of the wasted muscles are similar to that found in her sister.

No impairment of any form of sensation could be ascertained, nor is there any evidence of trophic disturbance save that her hands and feet are cold and cyanosed. The tendon-reflexes of the upper extremities are brisk and the knee-jerks much exaggerated and patellar clonus was easily obtained; but though the Achilles jerks are also greater than normal, there is no ankle-clonus. The abdominal reflexes are absent and the right plantar response was of the extensor type, but that from the left sole was indefinite.

These two cases are evidently examples of a progressive

family disease affecting at least two members of the same family, and possibly a cousin on the paternal side was afflicted with a similar or identical condition. The question naturally arises whether they conform to any recognised type or are merely casual deviations from the ill-defined class of hereditary spastic paralyses, in which the muscular wasting is a coincident symptom.

Similar cases in which several members of a family have been affected are recorded by other authors.

In the family described by Gee, the father and two of his children were similarly affected. The father never walked properly, and the difficulty in locomotion gradually increased, till, when he was examined at the age of thirty-seven years, he could get about only on crutches. His condition then closely resembled that of my two cases, but was more advanced. There was wasting of the hand muscles and spastic paresis of the lower extremities, with exaggeration of the deep reflexes.

His eldest child, a girl aged twelve years, never walked properly, and owing to progressive weakness of her legs, had to use crutches since she was seven years of age. Her legs were rigid, their tendon reflexes increased, and the small muscles of the hands wasted. His son, then aged eleven, had walked badly since his third year, and needed crutches since his fifth. His lower limbs were rigid and their deep reflexes increased; the arms were also spastic and the hands were held in a cramped position, but there was no obvious atrophy of their muscles.

Dr Ormerod's cases were three sons of a Jewish family of ten children. The eldest was twenty-three years of age when his condition was published. His gait has been affected since he was six years old, and since then walking had become more difficult. The hands had been weak for three years. His speech was natural. Nystagmus accompanied lateral movement of the eyes. The small hand muscles were much atrophied, and those of the legs uniformly wasted. Though the lower extremities were not rigid, his gait—he needed assistance—was spastic. The tendon-reflexes were exaggerated, and ankle-clonus and plantar extensor responses were obtainable.

His one brother, aged seven years, walked badly, the deep reflexes were exaggerated, and the plantar responses were of the extensor type, but his upper limbs were not affected. While in a third boy, then aged four years, there was in addition to these



symptoms weakness and diminution of the electrical excitability of the extensors of the wrists and fingers.

Maas has also recently published two cases, a brother and a sister being afflicted. In both the illness began at about the age of twelve years with weakness of the legs. The arms became weak and speech unnatural during the next few years. When examined the one was twenty-nine, and the other twenty-six years of age. The feet were in the position of pes cavus, the muscles of the lower extremities were wasted, and the limbs were weak and rigid. Walking was almost impossible. There was weakness of the arms with atrophy of their distal muscles, and the thenar and hypothenar eminences were absent. The facial muscles were paretic, and articulation was nasal. Sensation was unaffected, the tendon-reflexes were increased, and extensor plantar responses were obtained. The female patient was slightly demented, but her brother was fairly intelligent.

Hoffmann observed four more or less similar cases in one family. The affection first came to notice in childhood; it was probably congenital. Gait became progressively worse from the middle of the first decade of life, and in the second the hands, and later the forearms, wasted. Articulation was affected, and there was progressive psychical deterioration to imbecility. The wasting of the small muscles of the hands was extreme, and the fingers assumed the claw-position. The muscles of the lower extremities were also atrophied, more especially the distal ones, and the plantar arches had disappeared. Neither the upper nor the lower limbs were, however, rigid, though the knee-jerks were exaggerated and patellar clonus was obtainable in some of the cases.

Higier has published the cases of four sisters, each of whom began to walk badly in the eighth to tenth year. Paraplegia slowly developed with contractures and talipes equinus. Some three or four years later weakness and awkwardness of the hands set in. There was also in each case primary optic atrophy and mental defect amounting to imbecility. In all cases the musculature of the legs was small, and the hand muscles and some of those about the shoulder-joints were wasted in the elder patient, but not visibly affected in her three sisters, though their hands assumed unnatural positions.

Seeligmüller's cases were not quite similar. He describes amyotrophy associated with rigidity in four children out of a family of seven. The weakness became apparent in all towards the end of the first year of life and progressed gradually. The rigidity of the limbs was considerable, but the muscular wasting was more or less general. There was also affection of articulation and weakness of the lips, tongue and palate.

From these examples, and there may be many more in the literature, it seems justifiable to group such cases into a definite class, of which the family affection, the onset in early life, and the tendency to slow progression of the symptoms, are the distinguishing features.

The pathological basis of the disease has not been yet verified. Though the spastic symptoms may suggest the identity of the condition with cerebral diplegia, it seems advisable to separate the two conditions in terminology, as the slow progressive course of the malady through at least three decades of life and the frequent absence of marked mental symptoms are not characteristic of infantile diplegia.

Yet the occasional deterioration of intellect and the early appearance of spastic bulbar symptoms suggest that the lateral column affection which must be associated with the disease is of cortical origin rather than a primary fibre degeneration. There must be, too, a congenital defective vitality of some of the lower motor neurones, as the muscle-wasting is undoubtedly of spinal origin.

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**A NOTE ON ALCOHOLIC AND ARSENICAL NEURITIS.**

By ERNEST S. REYNOLDS, M.D. (Lond.), F.R.C.P.

**EVEN** at the present time, when we know so much more about arsenical poisoning than was the case before the epidemic caused by the accidental contamination of beer with arsenic in 1900, it is difficult to differentiate between the neuritis caused by alcohol and that caused by arsenic.

I have therefore been much interested in Dr A. Hill Buchan's valuable article on "Some Aspects of Alcoholism," published in this Review, for contrary to what he supposes, he to a large extent confirms my own remarks on some of the points of difference between these two forms of neuritis.

For many years previous to the epidemic occurring among beer drinkers in the north of England in 1900, which I discovered to be due to the presence of arsenic in the beer (*Brit. Med. Journ.*, Nov. 24, 1900, p. 1492), there had been such a large number of cases of so-called alcoholic paralysis in Manchester, principally among beer drinkers, that I had surmised there was at work some other factor besides alcohol, but what this was I could not guess until the contamination of brewing sugar with arsenic revealed that, at any rate during the epidemic, the neuritis was almost entirely arsenical in origin. But this accidental contamination would not explain the large excess (as compared with other medical centres) in Manchester of "alcoholic" cases during the fifteen years previously. These previous cases were on the whole so similar to those occurring during the epidemic (apart from certain skin eruptions) that it seemed possible that they might also be due to arsenic, and this possibility was made very probable when it was found early in 1901 that the malt used for beer-making in this district was largely contaminated with arsenic derived from South Yorkshire coke which had been used for years in the malt kilns. (See also the account in the "Proceedings of the Royal Commission on Arsenical Poisoning," vol. ii., of a serious outbreak of arsenical poisoning in Halifax in December 1901, proved to be due to arsenical malt.)

It was under these circumstances I said at the meeting of the Medico-Chirurgical Society in January 1901 (*Med. Chir. Soc. Lond. Trans.*, vol. lxxxiv.) that although I could not deny the

existence of true alcoholic neuritis, yet I was not convinced of it. Indeed, as Dr Hill Buchan says, I threw out the suggestion that all cases of so-called alcoholic neuritis might be really due not to alcohol but to arsenic. Many people have seemed to think from this mere suggestion that I deny the existence of alcoholic neuritis, but this I have never done either in speech or writings. Indeed as soon as it was possible after the arsenical epidemic was over, and as soon as I was able to see clearly out of the complications which the epidemic had caused, I stated at the Cheltenham meeting of the British Medical Association in August 1901 (*Brit. Med. Journ.*, 1901, vol. ii. p. 1044) that the epidemic had *not* proved that alcoholic neuritis did not exist, that records were in favour of true alcoholic neuritis, and that I could not bring myself to believe that every case of alcoholic neuritis recorded had really been caused by arsenic. Again, in my second examination before the Royal Commission on arsenical poisoning on March 7, 1902 (answer 8322), I related two cases which had been under my care suffering from true alcoholic neuritis, and I said, "My own opinion is that alcohol will cause peripheral neuritis."

Finally, in the *Brit. Med. Journ.* for July 25, 1903, p. 188, I stated that the cases of peripheral neuritis "occurring in pure spirit drinkers where there was no evidence of arsenical contamination are different from the cases of so-called alcoholic paralysis with which we (in Manchester, that is) have been so familiar for many years. They show some loss of power in the legs and arms, absent knee-jerk, paræsthesiæ of the feet and hands, only slight tenderness on pressure of the muscular masses, very little atrophy, no loss of memory and no œdema. Such cases are, moreover, considering the large amounts of alcohol consumed in this country, very rare." (The loss of memory I am willing to admit may occur in alcoholics.)

Further observations during the last eighteen months have strengthened me in this view. Previous to 1900 every case of peripheral neuritis occurring in alcoholics was commonly called alcoholic neuritis, and this was so whether the symptoms were severe or not. Now we are able to differentiate a little. In very slight cases of arsenical neuritis I doubt whether from the symptoms alone (skin lesions in such cases may be absent or very slight) it is possible to distinguish them from alcoholic neuritis,

for, to my mind, the appearances are very similar. I cannot agree with Dr Hill Buchan that in alcoholic neuritis the extensors of the wrist and flexors of the ankle are predominantly affected, and that arsenic appears to pick out and attack more severely the extensors of the fingers and toes. Indeed a reference to my photographs of arsenical neuritis (*British Med. Journ.*, Dec. 22, 1900) will show marked affection of the wrist and ankle muscles. Nor do I think that cramps are more often present in the feet than in the calves in arsenical cases, but rather the reverse. I think there is no doubt that many of the slight cases of so-called alcoholic paralysis seen in Manchester previous to 1900 (and still seen) were due to alcohol pure and simple, and these appear to me to be similar to the cases of true alcoholic paralysis occurring in Edinburgh and described by Dr Hill Buchan. But I think that nearly all the *severe* cases with marked paralyses and muscular atrophies, *great* pain on pressure of the muscles, well marked sensory symptoms (cutaneous hyperæsthesia, etc.), marked loss of memory for time and place and cardiac dilatation, seen in Manchester, were really due to arsenic. Such cases, previously to 1900 so common, are now rare, and when I see them I examine most carefully for slight pigmentation and keratosis, and generally find these symptoms, and at the same time get a history of beer-drinking (alone or in addition to spirits). Such a case, occurring in a woman, I have at present in my wards, with slight pigmentation round hair follicles and round the edges of old scars, and with keratosis on the feet. Professor Dixon Mann told me two days ago that within the last fortnight an alcoholic woman with severe peripheral neuritis came to see him at the hospital. He at once suspected arsenic, and from three ounces of her urine he obtained an abundant crop of crystals of arsenious oxide. Where the arsenic is coming from nowadays we do not at present know, but the cases still occurring rarely from time to time show how very susceptible some few people are to arsenic, and how important it is to remember the great probability of arsenical poisoning when the case is at all severe.

Other cases we see are more probably due to alcohol alone, and are, as I have said, apparently similar to those described by Dr Hill Buchan, but compared with our old-fashioned cases the symptoms are slight; there is seldom total loss of power of the

limbs or trunk, hardly ever any atrophy, the knee-jerks are not always absent, the muscular hyperæsthesia is not so intense, cutaneous hyperæsthesia is but slight, the memory is not so profoundly affected, there is but little or no œdema, no ataxia, and, of course, no skin lesions.

Finally, I am inclined to think that some of the differences of opinion concerning arsenical *versus* alcoholic neuritis expressed in different medical centres may perhaps be explained by saying that in Manchester we have seen much neuritis amongst alcoholics; most of the cases were probably arsenical and were severe, and accounted for our excess in numbers; the minority were due purely to alcohol and were comparatively slight in degree. Whereas in other centres possibly free from arsenic the few cases occurring were really alcoholic, and presented a more correct picture of true alcoholic neuritis (a comparatively slight affection) than we in Manchester, confused by arsenic, had been for years able to draw.

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## Abstracts

### ANATOMY.

**THE FORM OF THE NERVE ELEMENTS OF THE CERE-**  
(104) **BELLUM IN DIFFERENT VERTEBRATES.** (Ueber die  
Form der Nervelemente der Kleinhirnrinde verschiedener  
Vertebraten.) GURKOWITSCH, *Neurolog. Centralb.*, Jan. 16, 1905,  
p. 49.

THE conclusions of the author are based upon a study of the tissues of many different animals. The method used was the Golgi-Cajal.

Of all the cerebellar cortical elements the Purkinje cells alone possess an unusually large number of branching dendrites with gemmules. Those cells differ somewhat in different animals and vary in appearance at the various stages of development. The basket and Golgi cells apparently regress in their development with the growth of the animal. The higher the animal the more dendrites and gemmules do the Purkinje cells possess. This specialisation of form of the nerve cell corresponds to specialisation of function. It is possible that in the young animal the basket and Golgi cells are of greater importance than in the adult,

in which the nerve activity becomes centralised in Purkinje's cells. The author considers the dendrites and gemmules important factors in nerve activity.

DAVID OER.

# **CONCERNING THE NEUROGLIA OF THE OPTIC NERVES.**

(105) (*Ueber die Neuroglia des Sehnerven.*) JACOBY, *Klin. Monats. bl. f. Augenheilk.*, Feb. 1905, p. 129.

THIS paper is founded on Weigert's recent staining method for neuroglia, and specially deals with the arrangement of the fibrillary processes of the cells. Jacoby refers in the first place to the great difficulty of obtaining constant results by the method, but strongly recommends the use of a mordant, namely 5 per cent. copper nitrate, 2.5 per cent. fluorochrome, and 10 per cent. formaline without the addition of acetic acid, by which means he has obtained the specimens from which his most excellent sketches are made.

He believes that the fibrillary processes become quite distinct from the neuroglia cells, and that they run in bundles alongside the nerve fibres. He finds that they penetrate the pial sheath and become in this way intimately associated with fibres of mesoblastic origin. In the papilla the glia fibres are extremely delicate, and Dr Jacoby believes that they are largely responsible, firstly, for the appearance of a ring or half ring surrounding the entrance to the optic nerve and situated between the choroid and retina, and secondly, that they may account to a certain extent for the optical appearances of the physiological excavation in the papilla. The author devotes special attention to the arrangement of the fibrillæ at the angle where the nerve fibres pass into the retina and to the point of exit of the central artery from the disc. The paper is illustrated by some excellent sketches, and although the work is mainly of histological interest, the author promises to prosecute researches into pathological conditions by the same methods. One regrets that Dr Jacoby makes no reference to the question of the existence of different cells previously termed neuroglial, but on the other hand his paper is entirely confined to the fibrillæ.

ROBERT A. FLEMING.

# **THE NEUROFIBRILS ACCORDING TO THE METHOD AND**

(106) **RESEARCHES OF RAMON Y CAJAL.** (*Les Neurofibrilles d'après la méthode et les travaux de Ramon y Cajal.*) AZOULAY, *La Presse Méd.*, jan. 7, fév. 4, 1905, pp. 9 and 75.

THESE papers complete a series of communications relating to the new silver impregnation methods of Cajal for the exhibition of the

finer structure of nerve cell and fibre (the details of the technique will be found in the *Review of Neurology and Psychiatry*, 1904, pp. 146 and 293). They consist chiefly of an appreciation of the value of the method, and a demonstration of its applicability and significance. Azoulay agrees with Cajal's results in their entirety, and takes them as affording renewed proof of the integrity of the neurone hypothesis.

S. A. K. WILSON.

**A METHOD OF COLOURING THE NEUROFIBRILS BY MEANS  
(107) OF COLLOIDAL SILVER.** (Un metodo di colorazione delle  
neurofibrille mediante l'argento colloidale.) E. LUGARO,  
*Monitore Zoologico Italiano*, N. 11, 1904.

JORIS has recently described a method of producing a selective coloration of the neurofibrils by means of colloidal gold. The preparations are unsatisfactory, inasmuch as the colour imparted to the fibrils is too faint to admit of any conclusion being drawn as to their connections. Lugaro has found that colloidal silver may be used in a similar way, and that, with the aid of toning with gold chloride, it yields preparations in which the neurofibrils are deeply tinted and clearly differentiated from other elements. The details of the method are as follows:—

The best fixative for the tissues is one composed of 6 per cent. pure nitric acid and 10 per cent. formalin in water. It should be allowed to act for 24 hours. The pieces are then washed shortly in water and placed in 5 per cent. solution of ammonium molybdate in water for 24 hours, or for 36 to 48 hours when the smaller nerve cells are to be studied. They are next washed for a short time in water, rapidly dehydrated with alcohol, passed through chloroform and embedded in paraffin. Thin sections are fixed upon slides in the usual way. The paraffin is removed by means of chloroform, and the section is further washed with absolute alcohol, alcohol and water, and then with distilled water, in which the slides should be laid with the section downwards. This final washing must be very thorough (2 to 24 hours). The sections are then coloured by means of a 3 to 4 per cent. solution of collargol (manufactured by Heyden di Radebeul, Dresden), in which they remain for half an hour to one hour, the longer period and the stronger solution being employed when it is desired to obtain a view of the fibrils in the small nerve cells. The sections are next washed for a few minutes in distilled water and toned with a solution composed of .2 per cent. gold chloride, 1 part; 2 per cent. ammonium sulphocyanide, 1 part; and distilled water, 8 parts. This solution will keep for a considerable time. The slides are immersed in the solution, which should be kept in motion, until



the sections assume a grey or violet tint. Subsequent "fixation" in 2 per cent. hyposulphite of soda may be carried out, but is not absolutely necessary. Finally, the sections are thoroughly washed in water, dehydrated, cleared and mounted in balsam.

With this method Lugaro has made the following observations:—The intracellular neurofibrils are exclusively coloured by it. The medullated and non-medullated nerve fibres remain unstained. The axis-cylinder process is coloured only for a short distance after it leaves the cell, probably up to the point at which it becomes myelinated. All the nerve cells appear to contain a very fine reticulum, not revealed by any other method in use. No independent fibrils are visible. The impression of a longitudinal fibrillation results from the facts that the meshes of the network are elongated in certain definite directions, and that the trabeculæ running in these directions are specially thick. Even in the visible part of the axis-cylinder process the fibrils are not independent. The number of neurofibrils and the compactness of the reticulum that they form are such as to warrant the belief that the coloration of the elements concerned is complete. Even in the finest dendritic branches a structure of anastomosing fibrils is clearly recognisable.

W. FORD ROBERTSON.

### PHYSIOLOGY.

#### RESEARCHES UPON THE SPINAL LOCALISATION OF THE

(108) **MUSCLES OF THE PERINEUM AND RECTUM.** (*Recherches sur la Localisation spinale des Muscles du Périnée et du Rectum.*) S. IRIMESCO et C. PARHON, *Journ. de Neurol.*, Feb. 20, 1905, p. 61.

THIS attempt at determining the nuclear representation of the muscles of the perineum and rectum is based on the study by Nissl's method of the spinal cords of two patients who died with diffuse suppuration and gangrene of the perineum.

In the second and third sacral segments there is a noteworthy collection of small cells on the ventral margin of the ventral horn, known as group X of Onuf. The function of these cells is still unknown. The muscles of the perineum are represented in the lower half of the second and in the third sacral segments by a nucleus dorsal and median to this group, the cells of which are intermediate in size between those of Onuf's group and the ordinary motor cells. Another group of cells in the lower part of the third and in the fourth sacral segments placed median to the intermedio-lateral group, which is situated laterally at the junction of the ventral and dorsal horns, probably supplies the internal sphincter of the anus.

GORDON HOLMES.

## PATHOLOGY.

**RESEARCHES ON THE INFLUENCE EXERTED BY TRANS-  
(109) VERSE SECTION OF THE SPINAL CORD ON THE  
SECONDARY LESIONS OF THE SUBJACENT MOTOR  
CELLS, AND ON THEIR RECOVERY.** (*Recherches sur  
l'influence exercée par la section transversale de la moelle sur  
les lésions secondaires des cellules motrices sous-jacentes et  
sur leur réparation.*) C. PARHON et M. GOLDSTEIN, *Rev. Neurol.*,  
Feb. 28, 1905, p. 205.

IN this paper, the authors state the results of a number of experiments on dogs, cats and rabbits. In each one sciatic nerve was divided in the popliteal space and the spinal cord was also divided in the upper part of the lumbar enlargement at the same time. Control experiments were made in each case, one sciatic being alone divided. The multipolar cells of the posterior and post-postero-lateral groups were found to be specially affected as the result of the lesion of the sciatic. A very great difference is noted in the case of the animals with the cord divided and the control animals in which only the sciatic nerve was cut, the intensity of the cell change being much increased when all influences from the brain have been interrupted. The experiments on rabbits include animals which survived the double operation, 3 days, 11 days, and 23 days.

In the first pair of rabbits (3 days) the cells of the post-postero-lateral group of the rabbit with the cord divided were swollen, the nuclei also being enlarged. The cytoplasm of the cell is divided into two zones, of which the perinuclear is rich in chromatic substance, not altered in appearance to any extent, while the peripheral zone which is the larger of the two is clear and contains practically no chromatic granules, although a few fine granules may be seen near the periphery of the cell. Crossing this clear peripheral zone are protoplasmic processes which are rich in chromatic substances, while crossing the peri-nuclear zone are clear protoplasmic processes in which there is little or no colouring matter. These changes are not constant in all the cells of the group. In the control animal there is much less marked change, the cells being little swollen and the peripheral zone of the cytoplasm showing very slight and inconstant chromatolysis.

In the second pair of rabbits (11 days) the affected cells of the rabbit with the cord divided are markedly hypertrophied, the nuclei vesicular, the chromatic substance is rarified and fragmented especially at the periphery of the cell, but there is no zone with

special absence of chromatic granules. In the control animal which was killed at the twelfth day, the cells are larger than those on the normal side and there is less chromatic substance, but the changes are not very marked. The nuclei are vesicular and show a tendency to an excentric position.

In the third pair of rabbits (23 days) the affected cells of the rabbit with the cord divided show a large peripheral zone with marked chromatolysis and the central and perinuclear zone is paler. The cells are not much enlarged but the nuclei are swollen. The cells of both the posterior and post-postero-nuclear groups show these changes. In the control animal the affected cells are slightly hypertrophied and the chromatic substance more rarified, but they may easily be mistaken for normal cells and closely resemble those in the unaffected anterior horn.

In a pair of cats, in one of which the cord was divided at a slightly higher level, the animal lived 9 days. In the multipolar cells which correspond to the muscles of the leg and foot of the affected side, there was a diffuse chromatolysis with disintegration of the chromatic elements and swelling of the cells and of the nucleus. The authors noted the same changes occurred also in a dog.

It is thus evident that the section of the cord has a special influence on the cell changes which result from the division of their axis-cylinder processes, and that interference with the impulses from the cerebral centres favours the intensity of the alterations. In the rabbit after 23 days the alterations in the cells are very profound and greatly exceed the changes noted in the control animal.

The authors further refer to the effect of the suspension of the influences from the brain by dividing the cord on the process of repair of the motor cells after section of their axis-cylinder processes. For this purpose an experiment was made on a dog, one sciatic being divided and the cord being also cut through in the upper lumbar region. The animal lived 70 days. After this interval of time the process of repair should be complete if the section of the cord had been omitted. In the experiment it was found that the affected cells still showed a very marked degree of atrophy.

The authors point out that many more experiments are necessary before arriving at a final and definite result. They add a note as to the changes in the cells following a transverse section of the cord, and they refer to the researches of Van Gehuchten, Marinesco and others, but, as they themselves state, the experiments appear to be too few in number to make the observations of much authoritative value.

ROBERT A. FLEMING.

**CONTRIBUTION TO THE BIOLOGY OF NERVE DEGENERATION**  
 (110) (**RESULTS OF TRANSPLANTATION EXPERIMENTS**). (*Zur Biologie der Nervendegeneration [Ergebnisse von Transplantationsversuche]*.) L. MERZBACHER, *Neurolog. Centralb.*, Feb. 15, 1905, p. 150.

BESIDES the ordinary process known as degeneration, nerves may undergo necrobiotic change. This is characterised by absence of disruption of the myeline sheath, which is only slightly blackened by osmic acid, or only scattered granules may be stained black in it, and the surface of the fibres becomes coated by a coagulum. To microscopical examination such a fibre appears structureless, and it has lost its affinity to stains. Degeneration is an active retrogression, a vital process peculiar to the living fibre, while bionecrosis is a terminal change, signifying the death of the tissue.

The author's experiments consisted in placing isolated pieces of a peripheral nerve between the muscles of the same animal (auto-), or in those of an animal of the same (homo-) or of a different species (hetero-transplantation). In warm-blooded animals typical degeneration of auto- and homo-transplanted nerves resulted after some days. In cold-blooded animals the degeneration is much retarded, but the pieces of nerve still preserve the power to degenerate, and this change rapidly sets in when the temperature of the animal is raised. Degeneration only results when the nerve is transplanted into living tissue; only necrotic change occurs when it is placed in dead tissue or if it is kept in normal saline solution.

In hetero-transplanted nerves necrotic changes predominate or may alone be visible.

Consequently, in auto- and homo-transplantation isolated pieces of nerves live and remain capable of undergoing the retrogressive changes which are peculiar to living fibres, while hetero-transplanted nerves die, and are treated by the tissues into which they are inserted as foreign bodies.

GORDON HOLMES.

**THE PATHOGENESIS OF CHOKED DISC.** (*Ueber die Pathogenese (111) der Stauungspapille.*) A. SAENGER, Hamburg, *Neurolog. Centralbl.*, Feb. 1905, p. 98.

IN this paper the main theories regarding the origin of choked disc are shortly sketched. Dr Saenger supports the mechanical theory according to which the all-important etiological factor is the increased intra-cranial pressure. Against Leber's theory that the changes at the disc are primarily of an inflammatory nature, and

are due to the presence of irritants in the cerebro-spinal fluid filling the nerve sheaths, Dr Saenger adduces the well-known clinical facts that the most pronounced choked disc may occur in cases of intra-cranial tumours of small size, especially when lying in the posterior fossa, and also under various conditions in which it is not reasonable to suppose that any irritant could be secreted into the cerebro-spinal fluid, *e.g.* in intra-cranial aneurism, hæmorrhage into the optic nerve sheath, injuries to the skull. In support of the mechanical theory he cites those cases in which the choked disc has disappeared when the increased intra-cranial pressure has been relieved by a trephine opening. Even lumbar puncture may bring about a temporary diminution of the swelling of the nerve head. The occasional absence or spontaneous disappearance of choked disc in cases of cerebral tumour may in some cases be explained by closure of the communication between the sheath of the optic nerve and the subarachnoid space of the brain.

J. V. PATERSON.

**THE RELATION OF THE INTERNAL SECRETIONS TO EPILEPSY,  
(112) PUERPERAL ECLAMPSIA, AND KINDRED DISORDERS.**

CHARLES E. DE M. SAJOURS, *Journ. American Med. Assoc.*,  
Feb. 4, 1905, p. 364.

THIS paper is a continuation of the author's previous studies on the functional relationship between the adrenals, the pituitary body, and the thyroid. He believes that the metabolic processes of the body are sustained by all three glands acting simultaneously; he also agrees with those investigators who conclude that these glands are concerned with the destruction of toxic waste-products. From the very large number of facts adduced in his paper, he thinks one may justly contend that the pituitary body, the adrenals, and the thyroid jointly govern all oxidation processes, and, therefore, the functional activity of all organs. Thus Sajours argues that if the functions of the pituitary, the adrenals and the thyroid are interdependent—constituting what he calls the "adrenal system"—impairment of the functional integrity of any one of them must morbidly influence that of the others. Over-activity of the thyroid, for example, by surcharging the blood with this organ's internal secretion, will over-stimulate the pituitary body, and through them the adrenals, thus causing the symptom-complex of general hyper-oxidation—exophthalmic goitre. Conversely, lowering of the activity of the thyroid correspondingly lowers all oxidation processes, as instanced by the hypothermia of myxœdema, cretinism, etc.

The bearing of these various functions on the convulsive

disorders mentioned is next outlined, and the author believes that in these clinical states there are two sources of convulsions: the first, a toxic in the plasma circulating in the nervous elements; the second, excessive activity in the pituitary body, leading to a correspondingly marked oxidation in all organs, including the muscles. The first is certainly pathogenic, while the most that can be said of the second is that it is an exaggerated manifestation of normal functional activity, and, perhaps, protective.

In the therapeutic treatment of all conditions of toxæmia, attended with convulsions, it is rational to employ agents which tend powerfully to increase oxidation and general metabolism by enhancing the functional activity of the organism's protective system.

OLIPHANT NICHOLSON.

**THE PITUITARY AND PINEAL GLANDS, AND THE PERIPHERAL NERVES IN A CASE OF CRETINISM.** (Hypophysis, Epiphysis und peripherische Nerven bei einem Fall von Cretinismus.) BAYON, *Neurolog. Centralbl.*, Feb. 15, 1905, p. 146.

BAYON first gives a brief account of the normal histology of the pituitary gland and of the newer methods employed in such investigations. Following Benda's description, he divides the cells found in the anterior or glandular portion of the organ into three varieties, viz., chromophile cells with oxyphile granules, chromophile cells with amphophile granules, and colourless cells without granules. He agrees with Erdheim that the two former varieties of cell vary in number with age. Colloid substance is normally present not only at the junction of the two lobes, but also in the glandular tissue, the amount being greatest in old people; and in the posterior lobe, using Cajal's impregnation method, he finds numerous non-medullated nerve fibres. In the case of a cretin, 25 years of age, the chromophile cells with acid-staining granules were more numerous than normal, while the colloid material and the spaces which contained it were also increased. The connective tissue framework between these spaces did not contain glandular cells, and was somewhat thickened. There were no signs of degeneration or colloid change in the cells, but in the stroma were small granules believed by Erdheim to be fatty in nature, but which left a residue on extraction. Above all, the marked atrophic changes described by Ponfick as being found in the pituitary in a case of myxœdema were not found to be present.

With regard to the pineal body, Bayon found that the sand-like granules which are normally found in the adult organ were present only in very small numbers, and in this, as in several

other points, the condition of the gland resembled that found in the child.

No very definite changes were discovered in the peripheral nervous system.

W. E. CARNEGIE DICKSON.

#### CONTRIBUTION TO THE PATHOLOGY OF EXOPHTHALMIC

(114) **GOITRE.** (*Beitrag zur Pathologie der Basedowschen Krankheit.*) H. PÄSSLER, *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1905, Bd. xiv., p. 330.

INTRAVENOUS injections of a rabbit and dog with extract of a thyroid gland, obtained by thyroidectomy from a severe case of exophthalmic goitre, were followed by neither increased frequency of the pulse nor alteration of blood pressure. The writer consequently infers that the hypothetical poison contained in the diseased thyroid, and causing exophthalmic goitre, does not act on the circulation as a simple direct poison like a ptomaine.

W. T. RITCHIE.

#### THE PATHOLOGY AND BACTERIOLOGY OF ACUTE MENINGITIS.

(115) **GINGITIS.** WILLIAM T. COUNCILMAN, *Albany Medical Annals*, March 1905, p. 149.

THIS paper is one of a series on cerebro-spinal meningitis read before the Medical Society of the State of New York, January 31, 1905.

The three organisms most usually associated with acute meningitis are the diplococcus intracellularis, the pneumococcus and the streptococcus. All cases of acute meningitis are cerebro-spinal, and in all some extension of the inflammatory process into the brain tissue may be detected on careful examination, so that, according to Professor Councilman, they probably all deserve the term of meningo-encephalitis. The mode of infection may be through the blood stream, through the lymphatics, or by direct extension. The same character of exudation may be produced by all the organisms above referred to and even by the tubercle bacillus.

All cases of primary meningitis with rare exceptions are due to the diplococcus intracellularis. In sporadic cases of cerebro-spinal meningitis, it is possible that the infective agent may gain access through the lymphatics of the nose, for there is evidence to show that the diplococcus intracellularis may produce a rhinitis.

Of sixty cases of acute meningitis, eighteen were due to the pneumococcus, and eighteen to streptococcal infection, while

twenty-one cases were of the epidemic variety. Of the four remaining cases, two were staphylococcal; in the remaining two the etiology was not determined.

"In all the cases of epidemic cerebro-spinal meningitis examined, there was extension from the meninges into the brain substance and purulent infiltration around the vessels, sometimes accompanied with the presence of organisms, and an increase of the neuroglia of the cortex. In certain cases foci of diffuse gliosis were found in the white matter of the brain at a distance from the cortex. The disease has, further, a greater tendency to extend along the cerebral nerves than any of the other forms of meningitis." A form of pneumonia characterised by small foci of consolidation may also accompany the disease. The acute proliferative inflammation of the veins and arteries seen in pneumococcal and streptococcal meningitis was not found in the meningitis due to diplococcus meningitidis.

EDWIN BRAMWELL.

#### LESIONS OF THE RETICULUM OF NERVE CELLS IN RABIES.

(116) (*Trabajos del Laboratorio de Investigaciones Biológicas de la Universidad de Madrid.*) S. RAMÓN CAJAL and DALMACIO GARCÍA. From the Institute of Serotherapy and Bacteriology of Alfonso XIII., Madrid.

THE object of this work is to give in detail the singular metamorphoses experienced, during the infection of *Rabies*, in the reticulum of the various nerve corpuscles, especially as regards their constancy and diagnostic value, and their important signification in the study of the biology of neurofibrils. The skeleton of the neuronal protoplasm is the seat of interesting vital phenomena, of processes of reaction, growth and regeneration, worthy of careful analysis.

The preparations include three periods. 1. *Phase of paresis*, corresponding to the seventh and commencement of eighth day after inoculation into the brain of the rabbit. 2. *Phase of hemiplegia*, coincident with eighth day. 3. *Phase of total paralysis*, immediately preceding death, about end of ninth day.

Alterations in the spinal ganglia and in the trigeminus and vagus are but little accentuated on seventh day, only abundant during phase of total paralysis. In the initial phase the most significant change is hypertrophy of the superficial neurofibrils.

*Neurofibrillary hypertrophy* is seen often in cells surrounded by proliferated capsular elements (lesion of van Gehuchten), and is characterised by stout cords, in the cortical layer of the protoplasm intensely coloured and arranged in irregular meshes. The pres-



ence of delicate longitudinal striation in the cords makes one surmise that the lesion arises from fusion of several neurofibrils.

The writers give a detailed account of the changes found, but it may suffice to summarise that in the first phase, they found hypertrophy of the neurofibrils, partial and of fusiform aspect; in the second stage, a more marked degree of the same condition; and in the final stage, besides a further accentuation of this hypertrophy, vacuolation of the cells. This was found in varying degree in the different parts of the nervous system, but always in the same order.

The process of atrophy of the ganglionic cells by means of phagocytes shows three facts:—

1. The enormous resistance of the neurofibrils to the phagocytic aggression; indeed, they respond to the stimulation, becoming hypertrophied, and arranging themselves in dense fasciculi, and phagocytes are imprisoned in the cytoplasm, where they create vacuoles.

2. The capacity of the neurofibrils under stimulation to extend outside.

3. The resistance of the Axon. Even when the cells are in advanced atrophy, the axis-cylinder may subsist.

*Genesis and Signification of Neurofibrillary hypertrophy.*—In a former preliminary note two alternative explanations were given:—(a) Enlargement by dislocation and ameboid concentration of the material of the filaments. (b) Production of thick cords by means of fusion of numerous primary and secondary filaments. The authors now think that a hypothesis combining the two may meet with acceptance. They begin by putting forward their proposition that the neurofibrillary reticulum is not a mere inert conducting system, but represents a contractile apparatus having ameboid properties. Under normal conditions, the reticulum retains a fixed appearance; but under pathological influences, as inanition, anæmia, cold, etc., the neurofibrillary apparatus reacts, the chief anatomical expression being atrophy and disappearance of the secondary fibrils, their colourable matter concentrating in a few primary fibrils. In consequence of this concentration, great increase in size of these cords is found, and at the same time creation of large spaces, full of cellular fluid. From the partial and fusiform thickening, which is seen at the commencement, it would appear that the simple hypothesis of coalescence will not suffice.

The cordonal modification is an action of the living cell, since:—1. The hypertrophy coincides with the functional disturbances. 2. It is seen in neurons whose dendrites, axon and nucleus suffer no apparent change. 3. It is absent in corpuscles greatly altered, that is where the method of Nissl shows great

disorganisation. 4. In reptiles it is a normal phenomenon produced by cold. If the animal is warmed for several hours, the condition disappears. 5. It is seen, according to recent experiments, in embryonal or young nerve corpuscles, under the action of cold.

Three propositions are laid down:—1. The hypertrophy of the neurofibrils is related to the states of paresis, hypotonia, motor torpor, etc. 2. The atrophy and destruction of the reticulum, as also the alterations in the nucleus, coincide with paralytic symptoms. 3. The *lysic* process being a diffuse inflammation, the symptomatic interpretation is very complex.

As to the diagnostic value of neurofibrillary hypertrophy:—(a) The lesions just dealt with manifest themselves with absolute constancy in the nerve centres of animals affected with rabies, from the moment that the motor-sensory disturbances, and those of equilibrium, begin. (b) Moreover, the alteration in the framework of the cell was not met with in dogs and rabbits which had died of traumatic and infectious myelitis, diphtheria, tetanus, and those poisoned by phosphorus, arsenic and lead.

There are still many nervous diseases of animals to explore, and it would be premature to draw too decided conclusions, but the authors venture to assert that if, as they hope, this sign is wanting, even by the nitrate of silver method, in the pathological processes not analysed, it will serve as of great signification in determining the existence of hydrophobia in doubtful cases, and will compete, if not surpass, the lesions of Babes, of van Gehuchten, and of Negri.

The advantages of this new sign are: that it is found in all the nerve centres, and especially in those most attacked; that it is never wanting in hydrophobia (rabbit, dog and guinea-pig); that it does not exist in animals of advanced age (the sign of van Gehuchten is seen at times in decrepit dogs); and finally, that it constitutes a lesion, very evident and easily revealed by any of the methods recommended by one of the writers.

The authors recommend the two following methods from many:—

1. Rapid procedure. Pieces hardened three days in oven at a temperature of 28° to 40°, in nitrate of silver 1·50 per 100, are, after rapid washing in distilled water, submerged for 24 hours in the following reducer:—

Pyrogallic acid	.	.	1 to 2 grammes.
Water	.	.	90 cub. cent.
Formol	.	.	10 „

Wash two minutes in water, place for six hours in alcohol, and by simply fixing with warm scalpel on paraffin, can cut sections easily.

2. Method by alcoholic fixation. When time is not pressing, the following method is better. It has been described before:—

(1) Pieces not more than 3 mm. thick, 24 hours in absolute alcohol at 40°.

(2) Dry on blotting-paper and place in sol. of silver nitrate, 1.50 per 100, for four to six days in stove at 28° to 40°.

(3) Wash for a few seconds in dist. water, and submerge for 24 hours in—

Hydroquinone (or pyrogalllic acid)	.	1
Formol	.	10
Distilled water	.	90

(4) Absolute alcohol, embedding in celloidin, and moderately fine sections.

Choose in preference the following centres: spinal medulla, bulb, and central ganglia of the cerebellum. A. S. CUMMING.

### CLINICAL NEUROLOGY.

#### A CASE OF MYOPATHY OF THE ARAN-DUCHENNE TYPE WITH

(117) **AUTOPSY.** (*Un cas de myopathie à topographie type Aran-Duchenne suivi d'autopsie.*) DEJERINE and ANDRÉ THOMAS, *Rev. Neurol.*, Dec. 20, 1904, p. 1187.

THE patient, who died at the age of eighty, had presented during the last thirty-one years of her life a progressive muscular atrophy of the upper extremities beginning in the intrinsic hand muscles of the left side. The muscular atrophy was associated with fibrillary tremor but with no reaction of degeneration, and it never spread beyond the two upper limbs. After death a microscopical examination revealed no evidence of disease in the cord, the anterior roots or the peripheral nerves. On the other hand, the muscles affected showed marked changes of two kinds: (1) a simple shrinkage of fibres without loss of striation or increase of nuclei, and (2) marked atrophy with loss of striation, increase of connective tissue and proliferation of nuclei. There was extremely little evidence of fatty change. The author points out the extreme difficulty of diagnosis between muscular atrophy of myopathic and myelopathic origin in such instances as the one described.

E. FARQUHAR BUZZARD.

#### HYPERTROPHIC MYOPATHY IN THE SEQUEL OF TYPHOID

(118) **FEVER.** (*Myopathie hypertrophique consécutive de la fièvre typhoïde [dissociation des diverses propriétés des muscles]*). J. BABINSKI, *Rev. Neurol.*, Dec. 30, 1904, p. 1181.

A GIRL, who was seventeen at the time she came under observation, at the age of twelve went through an attack of typhoid fever

in the course of which she developed a monoplegia of the right upper extremity. The paralysis had improved somewhat during the year following the fever, but had since remained stationary. Under observation there was noticed a tendency to assume a position of adduction at the shoulder, flexion at the elbow, flexion of the wrist and extension of the fingers, and it was impossible for the patient to bring the arm, forearm and fingers into a straight line. The weakest movements were those of abduction of the thumb and extension of the wrist unless the latter was associated with flexion of the fingers. The great pectoral, deltoid, upper arm and anterior forearm muscles were hypertrophied, rather hard on palpation and more powerful than the corresponding muscles of the opposite limb, and there was a tendency for the same muscles to contract synergically in performing powerful movements with the left arm. The electrical reactions in the hypertrophied muscles were diminished to a slight extent, but did not show the characters of R. D. The reflexes were normal. The right face and leg showed no evident asymmetry at rest or in action.

In discussing the pathogenesis the author inclines to the view that the condition resulted from a peripheral rather than a cerebral lesion, and suggests that it was secondary to a myositis associated with some venous thrombosis, as in the cases recorded by Lesage and Cerné.

E. FARQUHAR BUZZARD.

**THE RÔLE OF THE SUPINATOR BREVIS IN MUSCULO-**  
(119) **SPIRAL PARALYSIS.** (L'action du muscle court supinateur dans la paralysie du nerf radial.) GUILLAIN and COURTEL-LEMONT, *La Presse Méd.*, Jan. 25, 1905, p. 50.

THE case recorded concerns an adult healthy male, 44 years of age, the conductor of an orchestra, over which he presides for three hours three evenings a week.

In July 1903 he noticed he could not move his right little finger with ordinary facility, and ten days later the ring finger became similarly affected. Five months later he found he was unable to extend the middle finger too. The condition has not altered since.

The functional impotence of these three fingers increases towards the ulnar side; when the hand is placed horizontally in the axis of the forearm, the little finger hangs vertical, the two terminal phalanges being slightly flexed; the ring and middle fingers are progressively less affected.

All the muscular movements of the limb from shoulder to finger, apart from those involving the muscles concerned, are unimpaired in range and power, nor are there any objective sensory changes, any trophic or reflex phenomena to be noted. The only

muscles which evince any electrical alteration are the extensor communis digitorum, and the extensor minimi digiti.

The explanation of this incomplete musculo-spiral paralysis is to seek. All ordinary toxic and other causes being excluded, the authors believe it stands in causal affinity to the frequent pronation and supination movements of the patient's arm in the exercise of his profession. Probably the posterior branch of the musculo-spiral has been injured as it passes through the belly of the supinator brevis. On pressure, pain is elicited over the point of emergence of this branch. A possible elucidation of the differing degrees of palsy of the three fingers affected, is the patient's custom of holding the baton more specially with the last two fingers of the hand.

The actual mechanism of the neuritis production, corroborated by dissection of various cadavera with this point specially in view, is probably the compression of the nerve between the superficial stratum of muscle fibres comprising the supinator brevis, and the bone, for the deep layer seldom descends so low as the superficial.

On the other hand, of course, it may be argued, on the analogy of certain musculo-spiral palsies due to actual tricipital contraction, that in the case under consideration the nerve branch may have been injured by the contraction of the supinator brevis.

Finally, the clinical correspondence between the condition detailed in this paper and drummer's palsy (*paralysie des tambours; Trommerlähmung*) is adduced as additional proof of the correctness of the opinion given above; and the view that the explanation of the latter disease is injury of the posterior branch of the musculo-spiral as it passes through the supinator brevis, is more satisfactory than any other hitherto forthcoming. In drummer's palsy the greater use of the extensors of the thumb establishes the incidence of the disease in these muscles.

S. A. K. WILSON.

**A CONTRIBUTION TO THE QUESTION OF GENERAL  
(120) PARALYSIS, TABES AND SYPHILIS. (Zur Paralyse-  
Tabes-Syphilisfrage.)** K. MENDEL, *Neurolog. Centralbl.*, Jan. 1,  
1905, p. 19.

THE first case is that of a boy, aged 10, who developed typical general paralysis at the age of  $8\frac{1}{2}$  years. His father suffered from tabes, his mother from general paralysis. Mendel refers to similar cases met with in the literature in which one or both parents of a juvenile tabetic or paralytic suffered from tabes or general paralysis.

Another case of juvenile general paralysis (and tabes) is described, in which there was, as in the former case, a history of parental syphilis.

A third case is mentioned, that of a man who developed tabes at the age of 67, having acquired syphilis at 51.

The late appearance of tabetic symptoms is of interest in relation to the late syphilitic infection, and, in the opinion of the author, affords support to the syphilitic etiology of tabes.

EDWIN BRAMWELL.

**CONCERNING THE RELATION OF TERTIARY SYPHILIS TO (121) TABES DORSALIS AND GENERAL PARALYSIS.** (Ueber die Beziehungen der tertiären Syphilis zur Tabes dorsalis und Paralysis progressiva.) CARL HUDOVERNIG und JOSEF GUSZMAN, *Neurolog. Centralbl.*, Feb. 1, 1905, p. 101.

THAT a very intimate relation exists between syphilis and tabes is the view held by the great majority of neurologists. Leyden, Goldscheider, and a few other observers of distinction, still, however, combat the existence of this relation, and one of the supports upon which they base their contention is the infrequency with which tabes is met with in the subjects of syphilis. Hudovernig and Guszman in this paper point to a conspicuous fallacy which occurs in statistics bearing on this question. For instance, in 563 of the 759 cases of syphilis recorded by Glaser, the date of infection was less than three years previous to the examination. In cases of tabes in which a syphilitic history is obtained, the disease does not usually develop until several years have elapsed after the date of infection, therefore in any enquiry it is necessary to take cases only in which some years have passed after the syphilis has been acquired.

In what proportion of patients suffering from tertiary syphilis are there signs of tabes and general paralysis? This is the problem which the authors have set themselves to solve.

For this purpose they have selected 50 patients from the Budapest Dermatological Clinic, suffering from undoubted tertiary syphilis (in 41 cases syphilitic skin lesions, in 8 cases syphilitic lesions of mouth, nose and pharynx were present), all of whom had acquired the disease at least three years previously.

The result of a careful examination of these patients for signs of tabes and general paralysis is expressed in the following table:—

Healthy nervous system	22 cases	44 per cent.
Combined system disease	1 case	2 „
Doubtful cases	4 cases	8 „
Tabes dorsalis	12 „	24 „
General paralysis	7 „	14 „
Tabo-paralysis	4 „	8 „

A table of the symptoms and signs in each of the above cases is given.

Since the examination was only made in reference to tabes and general paralysis, cases presenting hysterical and neurasthenic symptoms are included under the first heading. The doubtful cases were those in which symptoms suggestive of tabes or general paralysis were present, having developed after the syphilitic infection, but in which there was an absence of physical signs sufficient to justify a positive diagnosis of either of these affections. Excluding these cases, the very striking conclusion is arrived at that 46 per cent. of the patients had tabes, general paralysis or tabo-paralysis.

The authors take up also the question of the influence of hereditary predisposition to nervous disease in the production of tabes. In only 28 of their cases was a trustworthy history obtained. Among these they found that 64 per cent. of those patients who had a family history of nervous disease had tabes or general paralysis, while only in 41 per cent. of those in whom there was no such history were these diseases present; while admitting that the figures are small, the probable influence of hereditary predisposition receives suggestive support therefrom. They were unable to decide that the occurrence of a hereditary predisposition was of any importance in shortening the period between the syphilitic infection and the subsequent development of the tabes and general paralysis. Their conclusions regarding antisiphilitic treatment seem to show that the thoroughness with which the syphilis has been treated has little or no influence in preventing the development of tabes and general paralysis at a later date.

EDWIN BRAMWELL.

#### **TENDON REFLEXES AND SENSORY DISTURBANCES IN**

(122) **TABES DORSALIS.** (*Sehnenreflexe und Sensibilitäts-störung bei Tabes dorsalis.*) L. BREGMAN, *Neurolog. Centralbl.*, Jan. 1, 1905, p. 2.

THE observation of Babinski that the tendo-Achillis-jerk is frequently lost before the knee-jerk in case of tabes dorsalis has been confirmed by a number of observers. Kollarits examined 100 cases of tabes in reference to this point, and found that the tendo-Achillis-jerk was absent in 65, present in 30, and absent on one side in 5 cases. The knee-jerk was absent in 56 of these cases, present in 40, and absent on one side in 4 cases. In 11 cases the tendo-Achillis-jerks were absent, the other tendon jerks being unaffected. Sarbo, from an examination of 92 cases, obtained very similar results. In 5.4 per cent of his cases the tendo-Achillis jerks were lost, the knee-jerks being present, while in 2.1 per cent the reverse condition existed. Bregman briefly

describes three cases in which the tendo-Achillis-jerks were lost, the knee-jerks being present, and refers to the frequency with which objective disturbance of sensation, often undetected for the reason that it is not specially looked for, is met with in the distribution of the fifth lumbar and lower sacral roots. The tendo-Achillis-jerk, which, it will be remembered, is especially connected with the fifth lumbar and first sacral segments, is, however, often absent when no objective disturbance of sensation can be detected over the skin areas supplied by these roots; hence it would appear that the condition of the tendon reflexes is a more delicate indication of involvement of the spinal segments than is objective sensory disturbance.

EDWIN BRAMWELL.

**THE ABDOMINAL REFLEX IN TABES DORSALIS.** (Ueber den (123) Bauchdeckenreflex bei Tabes dorsalis.) QUINIO CATÒLA (Florenz), *Neurolog. Centralbl.*, Jan. 1, 1905, p. 7.

AFTER reviewing the literature of the subject under discussion, the author tabulates results arrived at from an examination of 38 cases, in 35 of which the disease was well advanced. In 9 cases the abdominal reflexes were active, in 9 cases normal, in 6 cases diminished, in 13 cases absent on both, and in 1 on one side. In 25 of the 35 cases above referred to, the abdominal reflexes were therefore still present. Conclusions drawn from these cases showed that the percentage frequency with which the abdominal reflexes were absent did not increase with the duration of the disease.

EDWIN BRAMWELL.

**A TROPHIC SORE ON THE PENIS IN TABES DORSALIS.** (Ein (124) Decubitus geschwür an Penis bei Der Tabes dorsalis.) ADALBERT VITEK, *Neurolog. Centralbl.*, Jan. 1, 1905, p. 17.

THE author describes the case of a patient suffering from tabes dorsalis in whom a trophic ulcer appeared on the under surface of the penis, apparently produced by irritation from constant contact with the neck of the urine bottle.

EDWIN BRAMWELL.

**VIRILE FUNCTIONS, FECUNDITY AND OFFSPRING OF (125) TABETICS.** (Facultés viriles, Fécondité et descendance des Tabétiques.) G. MILIAN, *Archiv. Gen. de Med.*, Jan. 10, 1905, p. 65.

THE author arrives at the following conclusions from an examination of 26 cases of tabes, in some of which the disease was advanced, in others in its early stages.



1. The majority of tabetics retain their *facultés viriles*. In a certain number these are diminished, erection is impaired, there is fatigue after coitus, etc.; very few are impotent. Impotence is observed especially in the cases in which there are bladder symptoms.

2. We possess few precise data as to the degree of fecundity of tabetics. Fecundity appears to be diminished in this disease, the proportion of sterile marriages being large.

3. As the same thing occurs in syphilis, it is very probable that the reduced fecundity depends on the syphilis rather than the tabes. The same argument may be applied to the high mortality which is seen in the offspring of tabetics.

4. The majority of the observations published as instances of hereditary tabes are cases of Friedreich's ataxia. There are, however, undoubted cases of hereditary tabes on record (observations of Remak and Babinski), but occurring in such a small proportion as to be almost negligible.

5. The small proportion of juvenile tabetics, the offspring of tabetic parents, in contrast with the large number of juvenile tabetics the children of syphilitic parents, points to hereditary syphilis as the direct cause of juvenile tabes.

6. The proportion of the children of tabetics suffering from nervous lesions of different kinds is large; but it does not appear that the tabes is directly responsible for this, since, firstly, hereditary syphilis produces the same predispositions and nervous lesions; and, secondly, in the tabetic marriages, the children born before the appearance of the syphilis and tabes show the same predisposition to nervous affections.

7. Although the children of tabetics are often *des tares nerveuses*, in other words have inherited the neuropathic predisposition which has been localised in the spinal cord of their progenitors, it does not seem that the subsequent development of tabes has notably increased the predisposition.

8. This last conclusion is not definite. Larger and more detailed statistics may perhaps modify it as the diagnosis of tabes advances.

EDWIN BRAMWELL.

**ON THE RETURN OF DEEP SENSATION, SPECIALLY BONE (126) SENSATION, IN TABES AFTER TREATMENT WITH EFFERVESCENT BATHS.** (Du Retour des sensibilités profondes et spécialement de la sensibilité osseuse chez les tabétiques par l'action des bains carbo-gazeux.) HEITZ, *Arch. gén de Méd.*, 21 fév. 1905, p. 449.

IN three cases of tabes treated with certain mineral baths, it was found that there was partial return of cutaneous æsthesia and more

or less complete of bone sensation. An explanation is the stimulation by the effervescent gas of peripheral sense organs, the effect reaching the posterior roots by some *action à distance*.

S. A. K. WILSON.

**A CASE OF MULTIPLE CEREBRO-SPINAL SCLEROSIS WITH  
(127) REMARKS UPON THE PATHOGENESIS OF THE AFFEC-  
TION.** F. X. DERCUM and ALFRED GORDON, *Amer. Journ. of  
the Med. Sciences*, Feb. 1905, p. 253.

AN account of a case of disseminated sclerosis is here given. According to the authors, this is only the seventh case of the disease with a pathological examination which has been published in the United States. They suggest that the low percentage of cases in America as compared with Europe may in part be accounted for by the small number of necropsies reported. The patient was a female, twenty-nine years of age, who three years previous to admission had fallen on the buttocks, since which time the symptoms had gradually developed. She had been admitted to the gynecological wards suffering from pelvic symptoms; cystic ovaries were diagnosed and an oophorectomy performed, after which she was transferred to the nervous clinic.

The patient was much emaciated. There was distinct atrophy of the thenar and hypothenar muscles, complete loss of power in the lower extremities, increased knee-jerks, ankle-clonus on the right side and a double extensor response. "Examination for sensation showed a hyperalgesia of the whole body." The joints, especially the shoulders and hips, were said to be painful. Very coarse intention tremor, scanning speech, lateral nystagmus. Pupils unequal and "responded to accommodation, but very little, if any, to light." Slight left-sided ptosis. Optic discs not examined. Incontinence of fæces and urine. Large bed sore over sacrum. Knee-jerks gradually disappeared. Shortly before death patient developed a profuse diarrhœa.

*Autopsy.*—Hypostatic congestion of lungs, chronic parenchymatous nephritis, pyelonephritis, cystitis and colitis. Brain deeply congested, pia arachnoid slightly cedematous. Beneath tentorium a large amount of clear straw-coloured fluid. Spinal dura distended with similar fluid. Upon microscopic examination, patches of sclerosis were found all through the cord and brain. The nerve cells were in many places preserved even in the midst of entirely discoloured portions. Degenerated fibres were present in the pyramidal tracts, but whether an independent condition or related to the sclerotic foci, the authors were unable to determine. Naked axis-cylinders were present in most of the diseased areas. Dilatation and thickening of the walls of the blood-vessels with leucocyte infiltration were present in places.

The authors allude to Eduard Mueller's view that disseminated sclerosis is in reality a multiple gliosis of the nervous system. They take up the position that all that it is possible at present to infer is that neither the nerve cells nor axis-cylinders on the one hand, nor the blood-vessels on the other, are primarily involved.

EDWIN BRAMWELL.

**THE COURSE OF THE SENSORY FIBRES IN THE SPINAL CORD,  
(128) AND SOME POINTS IN SPINAL LOCALISATION BASED  
ON A CASE OF SECTION OF THE CORD. MORTON PRINCE,  
*Journ. of Nerv. and Ment. Dis.*, Feb. 1905, p. 81.**

THE author describes a case of stab-wound of the cord between the sixth and seventh cervical segments. The chief symptoms were complete paraplegia and bilateral paralysis of the triceps and all movements of the fingers, with weakness of the wrist movements; on the *left* side tactile sensation was perfect, on the *right* there was profound anæsthesia of the whole lower extremity, trunk, and inner side of the upper extremity. The patient died four days after the injury.

The cord was found to have been divided from behind anteriorly, on the *right* side as far as a line running from a point corresponding to the anterior border of the pyramidal tract inwards to the central canal; on the *left* side to a greater extent, viz., as far as a line between the central canal and a point on the periphery still further forward and somewhat anterior to a point half way between the anterior and posterior roots.

The additional area cut on the *left* half includes (a) a portion of Gowers' tract, (b) a portion of the cerebellar tract, (c) a portion of the lateral ground bundle.

Since the whole of the posterior columns were destroyed, it is concluded that tactile sensations and probably pain impressions are conducted by paths other than the posterior columns, viz., the antero-lateral columns, and that this conduction is crossed.

But the posterior columns may also be a conducting path of sensation. The supposition of two possible paths of tactile conduction, one capable of taking up the function of the other in the case of a slowly-progressing lesion, is in agreement with the findings of Meyer, Petré, etc.

*Spinal localisation.*—The triceps centre is usually placed in the sixth, seventh and eighth cervical segments, and that of the wrist flexors entirely below the sixth. This case would indicate that the triceps centre is not above the seventh segment, and that the centre for the wrist flexors extends in part into the sixth.

References to the literature are given.

J. H. HARVEY PIRIE.

**A CONTRIBUTION TO OUR KNOWLEDGE OF THE DISSOCIATION  
(129) OF THE TEMPERATURE AND PAIN SENSATIONS IN  
INJURIES AND DISEASES OF THE SPINAL CORD. PRE-  
LIMINARY COMMUNICATION.** (Ein Beitrag zur Kenntniss  
der Dissociation der Temperatur und Schmerzempfindung bei  
Verletzungen und Erkrankungen des Rückenmarkes.) J. PILTZ,  
*Neurolog. Centralbl.*, March 16, 1905, p. 242.

THE author discusses the various views which have been advanced and are held by a great number of different authorities as to the intraspinal course of the fibres subserving thermal and painful sensations, and records conclusions deduced from the literature and from a case of probable hemisection of the cord by a stab wound, in which there was paresis of the leg on the same side with analgesia and thermanæsthesia of the opposite lower extremity. The following are the conclusions arrived at:—

1. Disturbances of temperature and pain sensations may be of cerebral, spinal or peripheral origin. We find them in hysteria, in capsular and cortical hemiplegia, in injuries and diseases of the spinal cord, and also in compression, injuries and diseases of the peripheral nerves.

2. Spinal thermanalgesia is met with in syringo-myelia, in traumatic lesions of the spinal cord, in compression of the spinal cord, in central hæmatomyelia and in hæmorrhage in the lateral columns of the spinal cord, in tabes, hypertrophic pachymeningitis, spinal syphilis, pressure myelitis and chronic myelitis.

3. Spinal thermanalgesia has a radicular cutaneous topography.

4. In the spinal cord a special tract exists for the conduction of temperature and pain impressions, which is anatomically distinct from that which subserves the conduction of tactile sensation and the muscular sense.

5. The localisation and course of the tract which conducts temperature and painful impressions is as follows:—posterior root, posterior horn, grey commissure, antero-lateral columns; ultimately the periphery of the same and in all probability Gowers' bundle.

6. The tract which conducts temperature and pain impressions consists of two neurons: the nerve cell of the first neuron (known as the protoneuron) lies in the spinal ganglion, the nerve cell of the second (known as the deutoneuron) lies in the grey substance of the contralateral half of the spinal cord.

7. A unilateral lesion of the grey matter of the spinal cord produces:—(1) A thermanalgesia on the same side, if it is limited to the posterior horn on the same side (*i.e.* the part through which the protoneuron passes). (2) A crossed thermanalgesia, if it destroys the grey substance in the neighbourhood of the anterior horn

(where the deuteronuron from the opposite side passes). (3) A bilateral thermanalgesia, if it involves the grey matter in both situations.

8. A lesion limited to the grey substance produces only a thermanalgesia, limited to a certain area of skin in the form of a hemizone, which corresponds accurately to the corresponding spinal segment which is involved.

9. A lesion of the lateral column, including the periphery of the same, *i.e.* Gowers' bundle, produces always a complete crossed thermanalgesia, from the toes upwards.

10. The hemizone of the homolateral thermanalgesia, which is produced by a lesion of the grey substance (the posterior horns), begins as a rule immediately below the situation of the lesion; and the contralateral, which results from a lesion of the grey substance in the neighbourhood of the anterior horn, begins about four vertebræ below the level of the lesion.

11. The upper border of the absolute crossed thermanalgesia, which is dependent upon a lesion of the white substance, or of the lateral columns, with the inclusion of Gowers' tract, lies about five vertebræ (or about five spinal processes) below the level of the lesion.

12. The upper border of the absolute crossed thermanalgesia, which is caused by a lesion of the peripheral part of the lateral columns, or of Gowers' tract, lies about 6-7 vertebræ (or spinous processes) below the level of the lesion.

13. If the upper border of the crossed absolute thermanalgesia only begins 6-7 vertebræ below the level of the traumatic lesion, then we can say with certainty, notwithstanding the presence of hemiplegia or paraplegia, that the pyramidal tracts are not cut through, and that the motor paralysis will improve and the patient will again be in a position to use the paralysed limbs.

14. In the region of the upper limit of the absolute crossed thermanalgesia (perhaps also on the same side) we often find a dissociation of warm, cold and painful sensations, the upper border of the thermanæsthesia being highest, that of the cold anæsthesia lowest, that of the analgesia between the two.

15. The dissociation of warm and cold sensations reach their maximum by the use of  $+50$  and  $0^{\circ}$ .

16. The upper limits of the thermanæsthesia for temperatures above  $+50^{\circ}$ —in other words, for  $+60^{\circ}$ ,  $+70^{\circ}$ ,  $+80^{\circ}$ , etc.—lie in sequence at a lower level below the thermal limit for  $+50^{\circ}$ , and approach the upper border of the limit for pain sensation.

17. The upper boundary for thermanæsthesia for temperatures under  $0^{\circ}$ —namely, for  $-5^{\circ}$ ,  $-10^{\circ}$ , etc.—lie in sequence from below upwards above the level, gradually approaching the upper limit of the analgesia.

18. The upper limits of the thermanæsthesia for temperatures between 0° and +50° lie between the upper limits for both these temperatures.

EDWIN BRAMWELL.

**POSTERIOR-BASIC MENINGITIS.** HENRY KOPLIK, *Amer. Journ. (130) of the Med. Sciences*, Feb. 1905, p. 266.

IN 1878, Gee and Barlow described twenty-five cases of non-tuberculous meningitis occurring in infants below two years of age. The essential feature in all their cases was "the holding back of the head." In 1897, Carr described a number of cases of hydrocephalus following meningitis. Still has made important contributions to our knowledge of the subject. In an article published in 1898, he stated that 49 cases of this uncommon and, in his opinion, sporadic disease had presented themselves during the previous ten years at the Great Ormond Street Children's Hospital. From the exudate in the ventricles and subarachnoid space in the more acute cases which he had examined, he had isolated a diplococcus closely resembling the diplococcus intracellularis of Weichselbaum and Jäger, but which he thought grew more luxuriantly in broth and on agar-agar and glycerin-agar. Still drew a distinction in this paper between epidemic cerebro-spinal meningitis and the posterior basic form occurring in infants which he describes, and pointed out that the former was the more rapidly fatal form, although they resembled each other in so far that in both the base of the brain was affected. In contra-distinction to suppurative vertical meningitis, complications are rare. In two out of fifteen cases described by Still there was an inflammation of the tendon sheaths. Barlow and Gee, writing in "Clifford Allbutt's System," showed that adhesions forming between the medulla and cerebellum may obliterate the foramen of Magendie and produce hydrocephalus.

The author of this paper "has never been able to establish until recently the fact, which was doubted by Still, that these cases may occur in epidemics of cerebro-spinal meningitis." In a recent epidemic occurring in 1904, he saw thirty cases of cerebro-spinal meningitis, eight of which were typical cases of posterior basic meningitis. Most of the latter cases were below two years of age. The previous history was negative in all; in one case there was a history of a miscarriage, and of another child having died some years previously of meningitis. In all the cases below two years of age with one exception, the onset was sudden, fever and vomiting being followed by rigidity of the neck and in some cases by convulsions. The children were emaciated, the limbs in some cases flexed, in some cases extended, head retraction present,

abdomen retracted or rigid. In some cases a tendency to cross the legs, in others purposeless movements of the upper extremities in a sort of arc in front of the face. Fontanelles bulging if still open. In some cases strabismus. No optic neuritis. Tâche cérébrale and evanescent erythema in all cases. Kernig's sign is of little value on account of the age of the patient, and because of the frequency of rigid flexor-contraction. The Babinski sign in most cases is absent. The temperature simulates that of tuberculous meningitis, often not rising above the normal until near the end of the case. Leucocyte counts in this series of cases were as a rule low, similar to what is seen in tuberculous meningitis, and therefore of no diagnostic value.

Lumbar puncture was not always successful in drawing off fluid. This was proved in some cases to be due to closure of the foramen of Magendie. In cases where the disease had lasted some weeks, lumbar puncture was always negative, the organisms probably having died out.

Of the six cases under two years of age, in three the meningococcus was found on lumbar puncture. In one of the remaining cases in which only a few drops of fluid were obtained by lumbar puncture, the meningococcus was demonstrated in the ventricular fluid post-mortem. In a second, apparently typical case no post-mortem was obtained. The third case had run a very chronic course, and although large quantities of fluid were drawn off, the examination of the fluid obtained by lumbar puncture was negative. In the chronic cases the mononuclear picture resembled what is seen in tuberculous meningitis.

Full records are given of the cases above referred to.

EDWIN BRAMWELL.

#### **SYMPTOMS AND DIAGNOSIS OF CEREBRO-SPINAL MENIN-**

(131) **GITIS.** HENRY L. ELSNER, *Albany Medical Annals*, March 1905, p. 152.

CEREBRO-SPINAL fever, a rare disease in Britain, is responsible every year for a large number of deaths in the United States. In the city of New York during the years 1894 to 1903 there were annually from 178 to 287 deaths registered as due to this disease. In 1904 the affection was met with in epidemic form, and no less than 1010 deaths were attributed to it between 1st January and 1st November. In the States, epidemics of cerebro-spinal fever have usually been characterised by the remarkable limitation of the ravages, the small number originally attacked and the high mortality. Pneumococcal meningitis is more virulent than is the form of the epidemic disease due to the meningococcus. Thus

Netler states that of 68 cases of pneumococcus meningitis, 61 died during the first days of the disease, while only one-third of the meningococcus cases died.

In Central New York the presence of other infectious diseases of the nervous system—acute anterior poliomyelitis and Landry's paralysis—during the prevalence of even limited epidemics of cerebro-spinal meningitis has been noticed. It appears that one attack probably gives immunity. Councilman found but five cases in which the disease showed itself twice in the same individual.

The author's experience as to the value of Kernig's sign in the diagnosis of cerebro-spinal meningitis is that the phenomenon is usually absent in cases in which the meninges are not involved, but that it is not always present with meningitis.

Friss of Copenhagen found Kernig's sign present in 88 per cent. of 60 cases of cerebro-spinal meningitis, Netler in 92 per cent. of his cases, and the author in 90 per cent. of the cases which he has observed. "The Kernig phenomenon is by no means pathognomonic of cerebral or cerebro-spinal meningitis."

The author enters into the question of lumbar puncture at considerable length, quoting the opinions of numerous observers as to its value. "Negative results will continue disappointing. Positive finds, when needed, prove exceedingly satisfactory."

Councilman and Eichorst are of opinion that so-called sporadic cases become foci from which epidemic cerebro-spinal meningitis may spread; if this is so, the importance of the evidence obtained by lumbar puncture in determining the diagnosis at once becomes apparent.

The author holds that "the pneumococcus as certainly causes cerebro-spinal meningitis as it does malignant endocarditis, and both of these fatal infections without pulmonary complications," and that "pneumococcal meningitis may follow distal infection."

Tubercular meningitis is to be distinguished by the long prodromal period, the common absence of leucocytosis, the frequent cranial nerve paralysis, the absence of other cases and the results of lumbar puncture.

EDWIN BRAMWELL.

**CLINICAL AND ANATOMO-PATHOLOGICAL STUDY OF THE**  
**(132) LACUNES OF CEREBRAL DISINTEGRATION.** (*Étude*  
*clinique et anatomo-pathologique sur les lacunes de désintégration*  
*cérébrale.*) CATOLA, *Revue de Méd.*, Oct. 10, 1904, p. 778.

It is a remarkable fact that a frequently occurring and macroscopically visible lesion in the cerebra of old hemiplegics was not recognised or described—at least, its significance was not appre-



ciated—till Marie's paper in 1900, the argument of which was further elaborated by his pupil Ferrand (see the *Review of Neurology and Psychiatry*, 1903, p. 809). The present communication emanates from the same *service*, and once more provides convincing evidence of the accuracy of Marie's original investigations, and the correctness of his interpretation of the facts. It is unnecessary again to detail the clinical phenomena characteristic of the condition, an indication of which will be found in the abstract above referred to. The bibliography of the subject is extensively scrutinised by Catola to show that cerebral lacunes are far from uncommon, though their import has not been seized; at the same time he amplifies the results obtained by Ferrand, and adds to the sum of our knowledge of a comparatively little known variety of vascular disease.

Among new clinical phenomena enlarged on in detail may be mentioned a peculiar deviation from the upright position evinced by the advanced *lacunaire*. He leans back, and will fall unless supported; his toes attempt to overcome the backward displacement by grasping the ground in exaggerated flexion (*orteils en griffe*).

Not the least valuable part of the present synthetic study of these lacunes is the discussion on their pathological anatomy, and their differentiation from cerebral porosis, *l'état criblé*, histological cerebral oedema, isolated dilatation round lenticular-striate vessels, etc.

The dependence of the lacune on arterio-sclerosis, and at the same time its rigid separation from that condition, are concisely set forth. In its essence it is due to a subacute or chronic inflammation of the perivascular lymphatic sheaths of certain cerebral vessels; it is the sequela of a subacute vaginalitis, which is microscopically revealed by leucocytic infiltration of the perivascular tissue.

The illustrations and bibliographical indications combine to render the present communication peculiarly complete.

S. A. K. WILSON.

#### ON A CASE OF BRAIN ABSCESS PRESENTING THE SYMPTOMS

(133) OF CATATONIA. (Ueber einen Fall von Hirnabzess bei katatonischem Krankheitsverlauf.) K. SCHMIDT (of Alt-Scherbitz), *Allg. Zeitsch. f. Psych.*, Sept. 2, 1904.

SCHMIDT reports the case of a young woman who began at puberty to show symptoms of mental disturbance; irritability, aversion to work, general mistrust, impaired memory. She felt ill, complained of sleeplessness, want of appetite, headache; lost flesh. Her condition gradually grew worse, she became depressed and con-

fused, and at the age of 23 made an attempt at suicide. Admitted to the asylum, she showed very variable behaviour: at times silly, frivolous, erotic, at other times quiet and mute. She showed marked negativism, impulsive actions, verbigeration: numerous hallucinations and delusions of persecution. At no period were there any nervous symptoms to suggest organic brain disease; no examination of the muscle sense nor of the fundus oculi was made; the diagnosis of catatonia was formed. Patient died of exhaustion at the age of 26 after being two years in the asylum. At the autopsy an abscess was found in the left superior parietal convolution of the size of a small hen egg, reaching nearly to the surface of the brain, but not down to the ventricle. The author gives his reasons for considering the abscess as the cause of the mental symptoms, and not a casual addition to an already existing psychosis.

C. MACFIE CAMPBELL.

**SOME THOUGHTS ON CONVULSIONS DURING INFANCY AND**  
 (134) **CHILDHOOD.** By HENRY ASHBY, *Lancet*, Jan. 21, 1905,  
 p. 135.

ALL through infancy the nervous centres are less under control than they are in later life. A special degree of this infantile susceptibility is characteristic of some families, just as a tendency to insanity or epilepsy is of others. This hereditary susceptibility is the most important predisposing factor in the causation of convulsions. An acquired predisposition is often seen in rickety children. It is a question, however, whether this is to be attributed to the rickets, or whether they are not both the result of chronic indigestion with absorption of toxins into the blood.

The exciting causes of convulsions in children may be divided into four groups. (a) *Reflex*, in which the stimulus may proceed from various parts, *e.g.* the gastro-intestinal tract (colic, etc.); the respiratory organs (bronchitis and whooping-cough); the middle ear; the nose and the mouth (dentition). (b) *Toxic*, as in the early stage of measles or influenza. (c) *Central*, as in meningitis or cerebral syphilis. (d) *Epileptiform*, in which no cause is found and the intellect is apt to be belated.

After dealing with the causation of convulsions, Ashby describes a few different types of the condition.

(A) Convulsions in new-born children are in his opinion far more frequently due to gastro-intestinal disturbance from artificial feeding, than to birth injury—an inflamed and overactive colon is often to blame. The short-cut cure for such cases is a wet nurse. In some instances the fits are due to intra-uterine degeneration or disease of the brain.

(B) The convulsions which set in *during teething* are far more frequently due to concomitant indigestion than to irritation from the teeth.

(C) Convulsions due to *organic intracranial disease* occur in meningitis, encephalitis, thrombosis, hæmorrhage, tumours and syphilis. They generally show nothing peculiar in their phenomena to distinguish them from toxic fits. There is, however, one type of convulsion occurring during infancy which is distinctly of the Jacksonian type. This is due to a syphilitic softening of the cortex of the brain. An infant a few months old who has suffered from coryza erythema and epiphysitis begins to suffer from slight one-sided convulsions. There is a momentary loss of consciousness or merely a dazed look. Contracture of the arm and leg affected follows and the infant shows signs of less and less intelligence. Post-mortem, a patchy softening is found in the cortex with degeneration of the arteries. Sclerosis seems to follow the softening.

The diagnosis between reflex convulsions and true epilepsy may be difficult, especially in children between 3 and 7 years old. If any obvious cause of reflex irritation is present, the prognosis is usually good. If such is absent, and especially if the mental state is peculiar, the outlook is generally grave. The occurrence of reflex convulsions shows a nervous constitution and indicates careful supervision during the rest of childhood.

Sometimes the convulsion is followed by more or less permanent damage to the brain from hæmorrhage or other lesion. In such cases there is usually high fever and prolonged unconsciousness. It is not easy to say under such circumstances whether the brain lesion is the cause or the result of the eclampsia.

Occasionally when the convulsions cease and the unconsciousness passes off the patient is found to be quite blind, although the pupils remain active and normal. This may last for weeks and then recover perfectly. It seems as if the visual area in the cortex were involved in the nerve-storm and remained for some time after in a state of exhaustion. Ashby has never seen a case of convulsions followed by permanent blindness. He records, however, an instance in which complete deafness occurred and persisted. Temporary aphasia with or without hemiplegia may occur. The pathology of all these complications is as yet very obscure.

In the *treatment* of the acute stage, subcutaneous injections of morphia form the most effective remedy. A strong infant of 6 months may have  $\frac{1}{16}$  gr. of this, and one of a year,  $\frac{1}{10}$  gr. Wasted, feeble, and newly-born infants should not be given morphia.

Chloroform is also useful, but its action passes off very quickly.

Chloral given by rectum acts more slowly, but its effects last longer. The bromides are too slow in action to be useful during the seizure. A dose of calomel is a very useful adjunct to the sedative treatment.

To prevent the continued and dangerous irritability of the nerve centres which predisposes so strongly to convulsions, close rooms, steam tents, etc., must be avoided, and the child allowed to have as much fresh air as possible. The nervous system should also be braced by cold salt douches, given after a warm bath every evening. Careful regulation of the diet is also important.

In the treatment of convulsions in older children, whether they are admittedly epileptic or possibly reflex, diet is of great importance. It should be of mixed quality and strictly limited as to quantity. It is not good to forbid meat altogether and allow excess of starchy food and sugar. Extras of all sorts between meals are to be forbidden.

As to school, the child should not be taken from school and allowed to run wild. "If 'running wild' means involuntary idleness, an aimless and empty existence without discipline and control and a more than ever concentration on self, then 'running wild' is the worst possible treatment for fits." Interesting occupation, mostly out of doors, is of great importance, and suitable companionship is very good for the child. The accurate performance of duties well within his power is to be insisted on, and is most wholesome. An active and fully occupied life is to be arranged for him.

#### **THE RELATIONS BETWEEN EPILEPSY AND MENSTRUATION.**

(135) MAURICE MAGUIN, *L'Echo Médical du Nord*, Dec. 25, 1904.

It is well known that some relationship exists between the function of menstruation and the occurrence of epileptic fits; but there are few observations which show such an intimate connection between the two states as to prove that they have made their first appearance simultaneously, and that thereafter every menstruation has been ushered in with epileptic seizures. For this reason the following case is of interest.

The patient, a single woman, thirty-one years of age, was always healthy. Her father, an old soldier, died six years ago of meningitis. He was a very nervous man, who indulged freely in alcohol, and readily gave way to violent outbursts of anger. The mother and the sister of the patient were both nervous women, but neither had ever suffered from epilepsy. The physiognomy of the patient herself is that of a degenerate, and her mental faculties are poorly developed. Her visual field is large. The cornea and

pharynx are insensitive; there is no cutaneous anæsthesia or hyperæsthesia present; the patellar reflexes are exaggerated; reactions intact.

She had no fits during infancy, but when five years old had violent fits of temper. At these times she developed a peculiar convulsive cough, in all respects resembling whooping-cough. When seventeen years old she had a great fright, and after that the first epileptic fit occurred. It was noted that she was then, for the first time, menstruating. Since then the fits always occurred at the menstrual periods. When twenty years old she became pregnant. The last periods before this were, as usual, accompanied by fits, but afterwards there was a complete cessation in the attacks, and throughout the whole course of pregnancy no fit occurred. The labour was normal and the child, a female, is now ten years old and healthy. On the reappearance of menstruation the fits returned, and afterwards each menstrual period came on with epileptic seizures, as was the case before pregnancy.

The seizures were typically epileptic in character; there was nothing to suggest that they were of hysterical origin.

The striking point is the very intimate connection between the epileptic attacks and the menstrual periods. Before the establishment of menstruation the patient, during her fits of violent anger, had a convulsive cough, which Maguin is disposed to regard as a form of masked epilepsy; no typical seizures occurred before the first menstrual period. The epileptic crises stopped entirely during pregnancy, and again reappeared at each menstruation—in this case they invariably accompanied menstruation. Hence the fits made their appearance in a manner as mathematical as the occurrence of the function of menstruation; and, as the author puts it, may be looked upon as a "cerebral discharge" accompanying the normal "genital discharge" of menstruation.

OLIPHANT NICHOLSON.

**THE NATURE AND TREATMENT OF EPILEPSY.** W. ALDREN (136) TURNER, *Lancet*, March 18, 1905, p. 706.

*Epileptic Equivalents.*—By epileptic equivalents are designated certain states of consciousness "differing not only from normal consciousness, but from the state of consciousness that usually occurs in epileptic attacks" (Peterson). The term really signifies a psychical state that in some degree takes the place of an epileptic seizure.

The following characters may be accepted as sufficient reason for regarding psychical clinical manifestations as epileptic equivalents: (1) the occurrence in the family history of similar or allied

conditions, interchangeable with forms of genuine epilepsy; (2) their association in the same person with the classical types of epilepsy; and (3) the existence of definitely epileptic characteristics, such as sudden onset, transitory duration, and irregular periodicity. Epileptic ambulatory automatism is the best known form of the epileptic equivalent.

The author believes that *some states of somnambulism in adults*, the condition of *narcolepsy*, and *some forms of migraine*, should be regarded as epileptic equivalents.

It is not legitimate to diagnose epilepsy unless there is evidence of loss or impairment of consciousness, although some incomplete manifestations may be unaccompanied by loss of consciousness.

*The Interparoxysmal Mental State.*—This condition is not wholly a direct consequence of the seizures, but is an expression of the same hereditary degenerative constitution which gives rise to the convulsions.

*Stigmata of Degeneration.*—Of one hundred epileptics who were examined as to the presence of structural stigmata of degeneration, seventy-five per cent. presented well-marked evidence of such. Of the two quinquennial periods in which the onset of epilepsy is most common, that from birth to five years is the most fruitful in the production of stigmata.

*Pathological Changes in the Brain in Epilepsy.*—Dr John Turner has found formation of coagula in the veins, capillaries and arterioles of the cerebrum and cerebellum of epileptics. The clots appear to be formed by the accumulation and amalgamation of blood plates. They contain phosphorus, probably as a constituent of their nucleo-proteid. These clots eventually degenerate and become absorbed.

The chief consequence of these thromboses is to deprive the cortical grey matter of the arterial blood necessary for its proper function and to produce a condition of blood stasis. If the coagulation and obstruction occur in the veins, as is commonly the case, the same result is brought about as if it occurred in the arterioles. The occlusion may be either partial or complete. If the latter, distension of the vessel proximal to the obstruction may ensue, with the result that small hæmorrhagic extravasations take place either into the cerebral tissue or the perivascular sheath. The nutrient artery of the cornu amonis is a common seat for thrombosis. The coagula are not passive processes occurring during a moribund condition, as they exhibit vital phenomena. Apparently, as a result of the deprivation of arterial blood, produced in the manner described, the large pyramidal or Betz cells show characteristic changes. Such are mainly seen in a large, swollen, clear nucleus.

The evidence that these thromboses do not take place in consequence of the convulsions lies in the fact that such thromboses are found in persons who have not necessarily died in status epilepticus, and it would also appear that unless the obstruction is complete, convulsions do not ensue.

The essential feature for the development of convulsions would appear to be *deprivation of arterial blood, coinciding with capillary and venous stasis, from motor areas*. The author accepts this view tentatively as a possible explanation of epileptic fits in general, from a consideration of some circumstances in which these seizures are prone to occur. First, the frequency of epileptic fits at night, especially during the hours of deepest sleep, when the cerebral blood pressure is at its lowest and capillary stasis is most likely to ensue. Secondly, the frequency of epileptic seizures in association with acute inflammatory diseases, such as pneumonia, enteric fever, and scarlet fever, in which there is an increased coagulability of the blood and a tendency to venous thrombosis. The puerperium also comes under this category.

*Treatment*.—The author thinks that the bromides are usually given in too large doses. If no benefit accrues from a daily dose of from 45 to 60 grains, some other remedy or method of treatment should be sought. Belladonna should be tried in all cases in which the bromides have failed.

The author's experience with Flechsig's treatment has not been satisfactory.

H. DE M. ALEXANDER.

**DISEASES OF THE NERVOUS SYSTEM SOMETIMES REGARDED  
(137) AS FUNCTIONAL, WITH PARTICULAR REFERENCE TO  
DIABETES.** W. HOWSHIP DICKINSON, *Lancet*, Dec. 10, 1904.

THE author is opposed to the somewhat wide use of the term functional in reference to nervous diseases. Hysteria, delirium and epilepsy may be called functional with less improbability than chorea, insanity and diabetes.

In diabetes, though we have not discovered the initial change we see sufficient signs of morbid action in the cerebro-spinal centres to indicate that they play a part in the disease. The extravasations which have been found are not large enough or constant enough to account for the diabetic process, but they suggest an attitude of the vessels which may have something to do with it.

These pathological indications which connect the brain with glycosuria are more significant when taken together with much that was previously known: the production of glycosuria in animals by puncture of the medulla, and in man by wounds and

injuries affecting the same part of the brain, and the occurrence of diabetes in consequence of mental emotion in the shape of grief, terror or anxiety. The temporary exaggeration of diabetic diuresis under mental perturbation is not uncommon, and Dr Smith and Mr Page have shown that engine-drivers, probably on account of the anxious nature of their calling, are especially liable to diabetes.

If insanity be a disease of the brain, and glycosuria, as with Bernard rabbits, a result of cerebral irritation, the two should sometimes concur. The author examined 106 insane patients, and found glycosuria in 18 cases. Dr Bond found glycosuria in 5.32 per cent of recent cases of insanity, and it is more especially frequent in patients with melancholia. Dr Maudsley has shown that diabetes and insanity "are certainly found to run side by side, or alternately with one another, more often than can be accounted for by accidental coincidence or sequence."

Coming now to the post-mortem condition of the brain and cord in diabetes; as with tetanus and chorea, we find results of morbid relation between the blood-vessels and their surroundings which, however interpreted, do not admit of dispute. We find perivascular hæmorrhages, not by rupture, but exudation, enlargement of the perivascular spaces giving in section and in parts a cribriform appearance, a dilatation of the central canal of the spinal cord, and, as has long been known, the liver is found congested. Abnormal these changes certainly are, but none of them are peculiar to diabetes, nor with diabetes are they constant in place or degree. As with tetanus and chorea, they mainly indicate hyperæmia.

We have much to learn about gout and heredity, and about the origins and kinds of diabetes; but, whatever the future may have in store for us, it can never abolish the experimental and clinical facts which associate glycosuria with morbid influences acting by way of the nervous system, nor dissociate diabetes from certain disorders undoubtedly pertaining to the same system which, notwithstanding that they were, or still are, by some regarded as "functional," display signs of perivascular disturbance which forbid them to be so regarded if by functional is meant "without material change."

H. DE M. ALEXANDER.

**MIGRAINE A BULBO-PONTINE SYNDROME, DUE TO VARIOUS**  
(138) **CAUSES.** (*La Migraine commune, Syndrome bulbo-protubérantielle à étiologie variable.*) L. LÉVI, *Rev. Neurol.*, Feb. 15, 1905, p. 166.

**THERE** is no satisfactory explanation of the symptoms of migraine. The hypotheses most generally accepted are, that it is due to a



spasmodic (Dubois-Reymond) or paralytic (Möllendorf) affection of the sympathetic system, or that it is a neuralgia of the trigeminal nerve (Brissaud); but none of these suffice to explain all symptoms.

Lévi assumes the existence of a migraine centre in the floor of the fourth ventricle. The trigeminal fibres which supply the dura mater may be represented there, and the hemicrania would be due to a discharge from them. The discharge may spread to the neighbouring nuclei and give rise to the various other symptoms: to the glosso-pharyngeal (nausea), vagus (vomiting and modification of pulse and respiration), to Deiter's nucleus (vertigo), and to the vasomotor, salivary, urinary and ocular centres. The spread of the discharge may be limited, so that only some of these centres be affected. It may also spread to the facial nucleus, causing spasm or palsy of the face.

This migraine centre admitted, the attacks depend on the susceptibility or instability of the centre, and on direct excitation of it. The instability of the centre is often hereditary, but may be acquired, as each attack makes the occurrence of subsequent ones easier.

The direct excitation may be any emotional or visceral disturbance, but is generally an auto-intoxication. The attacks of migraine which occasionally accompany the menstrual periods in woman may be due to auto-intoxication of ovarian origin.

The author suggests that an attack of migraine may be a defensive process, a method of getting rid of the toxic substances; thereto is due the marked *bien-être* which generally follows an attack.

GORDON HOLMES.

#### **A CASE OF HEMIPLEGIA WITH CONJUGATE DEVIATION OF (139) THE HEAD AND EYES IN A PERSON BORN BLIND.**

(Un Cas de Hémiplégie avec Déviation conjuguée de la Tête et des Yeux chez une Aveugle de Naissance.) J. DEJERINE et G. ROUSSY, *Rev. Neurol.*, Feb. 15, 1905, p. 161.

A WOMAN, aged 71 years, who had been blind from birth owing to an attack of purulent ophthalmia, suddenly developed left hemiplegia, with conjugate deviation of the eyes and head to the right. The eyes were freely movable on volition, but the head was held inclined and rotated to the right by tonic muscular spasm, which offered considerable resistance to passive movement. She died ten days after the onset of the attack. The hemiplegia was due to softening in the posterior limb of the internal capsule, but it was most intense in the retrolenticular segment, where it destroyed the optic radiations.

The chief interest of the case lies in its bearing on the question of the causation of the deviation of the head and eyes from the paralysed side. According to one hypothesis, that of Bard, Roux and Dufour, the paralytic deviation is always associated with hemianopia, and is of sensorial origin, *i.e.* it is an active subconscious movement, due to the relative overaction of the sensoriomotor centres of the healthy hemisphere; while Grasset, in part accepting this, assumes in addition the existence of an oculomotor centre in the region of the angular gyrus, the destruction of which results in paralytic deviation of the eyes to the side of the lesion.

The authors conclude that (1) as the case they report was blind from birth, and as her visual centres were consequently uneducated and inactive, the deviation of the head and eyes cannot be due to sensorial defect; and (2) the deviation of the head and eyes is not a purely paralytic phenomenon, as in their case it was maintained by tonic spasm of the muscles of the healthy side.

GORDON HOLMES.

#### **BI-TEMPORAL HEMIANOPIA AND DIABETES INSIPIDUS.**

(140) (*Bitemporale Hemianopsie und Diabetes insipidus.*) E. REDSLOB (Bern.), *Klin. Monatsbl. f. Augenheilk.*, Vol. xliii., Bd. i., p. 226, Feb. 1905.

THE author divides cases in which the two above conditions are found combined into two groups—those caused by cerebral tumour and those caused by trauma—and he describes the case of a girl, aged 14, who was under his care suffering from diminution of visual power following upon fracture of the base of the skull occurring three months previously. The eye condition was accompanied by headache, great thirst, and by the passage of an abnormally large amount of urine, and on examination the right pupil was found to react to light much more sluggishly than the left. Contraction for accommodation and on convergence were normal on both sides. The hemianopic pupillary reaction varied, being present on some days and weak or absent on others. Fundus normal.

Complete bi-temporal hemianopia was found, the field of vision being sharply cut off at the middle line. The nasal parts of the field were perfectly normal in both eyes, and the condition was presumably due to rupture of the optic commissure, while the diabetic condition, though it might be due perhaps to some injury to the floor of the fourth ventricle or to the medulla, might, on the other hand, have no direct connection with hemianopia—might, in fact, be a “neurose due to trauma” following the head injury.

W. E. CARNEGIE DICKSON.

**A CASE OF AMNESIC APHASIA.** ("Aphasie Amnesique.")

(141) A. HALIPTÉ (of Rouen), *Nouv. Icon. de la Salpêtr.*, Jan. 1905, p. 36.

**AFTER** remarking upon the rarity with which pure examples of the classic types of aphasia are met with clinically, the writer draws attention to those cases which have been classed by Pitres under the term "amnesic aphasia." Following Pitres he defines amnesic aphasia thus:—"Every patient who understands what is said to him, who can read aloud, who can pronounce and write perfectly such words as he can recall and who is only prevented from pronouncing or from writing other words by the fact that he cannot recall these words at the opportune moment, suffers from pure amnesic aphasia."

There are three varieties of amnesic aphasia:—

- (1) The inability to recall chiefly concerns the substantives. Periphrases are used to replace the substantive of which the recall fails. To this variety the term "autonomasia" has been given by Luys.
- (2) The memory of sentence construction is lost. This variety is called "agrammatism."
- (3) This variety is met with in polyglots who lose entirely the memory of a language which they have learnt, while other languages can be used fluently.

A case of the first variety combined with word deafness is reported in great detail and the pathological condition is excellently demonstrated by five block figures and three diagrams.

The following is an abstract of the most important of the clinical and pathological details.

Autonomasia, paraphasia, word blindness, partial letter blindness, paraphagia both for spontaneous writing and for dictation.

At the autopsy an area of softening was present upon the external surface of the left occipital lobe. Commencing at the pole of the left occipital lobe it stretched forward, involving the upper part of the angular gyrus, the first parieto-temporal convolution; the second parietal convolution was slightly involved but the posterior extremity of the second temporal convolution was not involved. There was destruction of the underlying white matter including the superior longitudinal bundle and the vertical occipital bundle of Wernicke.

There was no involvement of the following regions: the first and second temporal convolutions, the cuneus, the lingual and fusiform lobes, the optic radiations.

In addition two small foci of softening were present, one in the left paracentral region and the other in the sub-cortex of the foot of the right third frontal convolution.

The author refers to similar cases, details of which have been published by Guido Banti, Paul Serieux and Bianchi. He concludes that the inferior parietal lobule, including the angular gyrus, is the conclusive centre for the recall of word memories.

A sub-cortical lesion produced a similar syndrome in a case recorded by Trenel, and Pitres himself considered amnesic aphasia to be the result of lesion of the sub-cortical association paths. The author inclines to the hypothesis of Pitres. He points out especially the favourable course as regards speech recovery in amnesic aphasia, and for this reason, if for no other, correct diagnosis is of importance.

JAMES COLLIER.

**AMUSIA** ("Les Amusies.") G. MARINESCO, *Semaine Médicale*, (142) fevr. 1, 1905.

THIS discourse treats of certain troubles both upon the receptive and upon the executive side of the musical faculty which are broadly similar to those defects of the speech faculty which are grouped under the name "aphasia."

It commences with a history of the subject, and shows that while Strumpf and Stricker in Germany were the first to formulate ideas upon the subject, the great part of our knowledge resulted from the labours of G. Ballet in France, and of Oppenheim, Knoblauch and Wallaschek in Germany who demonstrated the reality and independence of the motor images arising in the mouth, larynx and respiratory muscles during singing.

Kast demonstrated upon a patient of his the complete separability of amusia and aphasia. Ballet contrasts a comparable variety of amusia with each classic form of aphasia. Brazier, writing in 1892, distinguishes the following primary types of amusia: motor amusia, music deafness, music blindness. Motor amusia may be vocal or instrumental. Other important workers upon this subject have been Paul Blocq, Brissaud, Edgren, who collected 52 cases, Donath and Würtzen.

The following case is then reported in detail:—The patient was 47 years of age, and was professor of the bassoon. He was seized with right hemiplegia, and was unconscious for some hours. The hemiplegia rapidly diminished, leaving the slightest degree of right hemiparesis. Speech was only affected in that he sometimes had difficulty in recalling a word. Spontaneous writing was slightly affected. He could not write music spontaneously. He could not transcribe from memory airs that he sang well. He could write music from dictation much better. He could copy music well, but not so quickly as before his illness. He had no

word-deafness nor music-deafness. He at once recognised intentional faults played upon the mandoline, even to the half tone. Spontaneous singing was impossible, but if someone else sang the first few notes of a song he knew he joined in and sang correctly enough so long as the other person continued singing.

When attempting to play his bassoon he made many mistakes with both hands, but especially with the right hand. To sum up, this patient presented incomplete motor amusia, involving both vocal and instrumental music; in addition, musical agraphia was present.

The writer proceeds to contrast the case with cases of sensory amusia which have been recorded by Charcot, Proust, Finkelnburg, Bernard, Brazier and by Dejerine.

The author concludes that the motor centres for musical language are located in the motor area of articulate speech. He suggests that the lower part of the left third frontal convolution is particularly concerned with musical language, the upper part being chiefly concerned with articulate language.

In the treatment of such cases persistent attempts in re-education are advocated.

JAMES COLLIER.

#### CONTRIBUTION TO THE STUDY OF THE CONNECTION (143) BETWEEN APHASIA AND MENTAL DISTURBANCE.

(Beitrag zum Studium über den Zusammenhang von Aphasie und Geistesstörung.) O. ALBRECHT (of Graz), *Allg. Zeitschr. f. Psych.*, Nov. 1904.

FOR practical purposes Albrecht divides the combinations of aphasia and psychosis into three groups: firstly, the two may be connected merely through the fact that they are both due to the same cause, as in cases of general paralysis, epilepsy, etc., presenting aphasic symptoms; secondly, the aphasia may develop out of the mental disease as in Wernicke's case, where a transcortical motor aphasia developed from a motility-psychosis; thirdly, the psychosis may result from a previously existing aphasia. Most cases in the third group show defect conditions; the author calls attention to other forms of psychosis resulting from aphasia. He gives two very complete observations of patients with aphasia of sensory-motor character, who several months after the onset of the aphasia began to have hallucinations and to develop delusions of a more or less systematised nature. He emphasises the influence of the painful effect caused by a difficulty of expression and understanding of which the patient himself is conscious, on the development of the paranoic condition; the hallucinations are correlated with the destruction

of paths of association, causing irritative phenomena in sensory fields. The psychosis may thus be looked on as developing upon the basis of altered cerebral functioning and under the influence of a painful effect and hallucinations, all of which factors depend upon the aphasia. Albrecht groups with his two cases, two cases of Cramer and Kreyer, where a paranoic condition developed out of a sensory aphasia. In conclusion, he calls attention to the value for psychiatry of the thorough examination of the mental status of aphasics.

C. MACFIE CAMPBELL.

**THE TREATMENT OF APHASIA BY TRAINING.** CHARLES K. (144) MILLS, *Journ. Amer. Med. Assoc.*, Dec. 24, 1904.

THIS paper is a resumé of the measures which have been adopted for the re-education of aphasic patients. Some results of this treatment are given in detail.

The methods advocated are those of Wyllie and Goldscheider, and may be summed up as follows:—Repetition after others; reading aloud; copying; writing from dictation; the use of the phonetic alphabet and of phonetic readers; the employment of vision to aid in the imitation of the movements of articulation, enunciation and vocalisation as made by others. Retraining in grammar where agrammatism exists; the repetition of phonetic syllables.

JAMES COLLIER.

**PSYCHIATRY.**

**THE EARLY FORM OF DEMENTIA PARALYTICA.** (Frühform der (145) *Dementia paralytica*.) G. EISATH, *Monatssch. f. Psych. u. Neur.*, Dec. 1904.

EISATH reports the case of a young woman, aged 23, who in the spring of 1900 slept almost uninterruptedly for two weeks; later in the year she became greatly elated with occasional fits of depression and irritability, and was admitted to the psychiatric clinic in October 1900. Her mental condition suggested the diagnosis of manic-depressive insanity; no physical symptoms of importance were observed. Patient made a good recovery and was almost normal when discharged in June 1901. She remained at home a year, showing only slightly defective intelligence and impaired memory.

In May 1902 she again became elated; on readmission to the clinic it was observed that the right pupil was irregular, wider

than the left, and did not react so well; otherwise her physical condition was as before. For several months she was elated with intervals of depression, with occasional outbursts of anger and obscene abuse. In September a series of catatonic symptoms developed and persisted for about two and a half months; patient then began steadily to improve and was about to be discharged, when she suddenly had a series of epileptiform convulsions and died in February 1902.

Examination of the brain disclosed the macroscopic and microscopic appearances characteristic of general paralysis of the insane. The author calls attention to the difficulty of diagnosis from dementia præcox before the terminal convulsions, in the absence of any history of syphilis inherited or acquired, and of any marked physical symptoms save those of the right pupil.

C. MACFIE CAMPBELL.

#### **SYMPTOMS OF APRAXIA IN A CASE OF SENILE DEMENTIA.**

(146) (*Apraktische Symptome bei einem Fall von seniler Dementia.*)

H. MARCUSE (of Dalldorf), *Centralbl. f. Nervenheilk. u. Psych.*, Dec. 1904.

MARCUSE gives the case of a patient, aged 60, with symptoms of transcortical motor aphasia, poor orientation for time, place and person, markedly impaired memory for the remote past and facts of general experience, and extremely poor retention of new impressions. The interesting feature in the case consisted in her reactions to simple orders; *e.g.* when told to put out her tongue patient failed to do so, and made inappropriate movements, but was able to lick a stamp, stretching her tongue well out. She made quite inappropriate movements when asked to point with her forefinger, but if asked to point out a picture did so correctly. The orders were well understood, and the motor apparatus not affected.

The author suggests as explanation for some of the apractic symptoms the extremely poor retention of the patient, causing the fading of the idea of the action to be performed before all the steps were carried out. For those inappropriate actions where the short time-interval made such an explanation impossible, or where it was a case of imitating a movement shown, he suggests the explanation that the representation of the action to be carried out had too little interest to give it the force necessary for an appropriate response: thus the patient could thread correctly a needle, although unable to imitate a military salute. This diminished power of association would have for its basis the senile atrophy of the brain.

C. MACFIE CAMPBELL.

**KORSAKOFF'S SYMPTOM-COMPLEX IN ITS RELATIONS TO  
(147) THE DIFFERENT FORMS OF DISEASE. (Der Korsakowsche  
Symptomenkomplex in seinen Beziehungen zu den verschie-  
denen Krankheitsformen.) BONHOEFFER (of Heidelberg), *Allg.  
Ztschr. f. Psych.*, Sept. 2, 1904.**

BONHOEFFER in this paper, read before the Deutscher Verein für Psychiatrie, considers it advisable to use the term Korsakoff's symptom-complex only where there are the four symptoms—extremely poor retention, defective memory for the recent past, disorientation and confabulation. He first discusses its relation to chronic alcoholism; here the syndrome is preceded in the great majority of cases by delirium tremens, in other cases by a stuporous condition almost invariably accompanied by neuritis, and occasionally by asymbolic and aphasic symptoms. While in non-alcoholic cases the syndrome may also be ushered in by a delirium, this delirium is different from that of the alcoholic. In all cases of polioencephalitis hæmorrhagica superior which the author examined, he found neuritic symptoms and the amnesic complex; they were therefore examples of the psychosis described by Korsakoff. The symptom-complex is found in 3 per cent. of chronic alcoholics, and much more frequently in women than in men; complete recovery is rare, but great improvement is not uncommon. Chronic alcoholism alone is insufficient to cause the occurrence of the syndrome; there is always an additional toxic cause.

Acute infectious diseases, even on a non-alcoholic basis, may lead to Korsakoff's symptom-complex, which is ushered in by a delirious or stuporous condition; chronic poisoning by lead and arsenic are also causes, and in the latter case the prognosis is more favourable. Senile and arterio-sclerotic changes often produce the syndrome in a very pure form; here it is preceded by apoplectic or minor attacks, and is accompanied by emotional dulness. Bonhoeffer has never seen it develop out of the ordinary senile memory defect without intercurrent acute symptoms. In senile and arterio-sclerotic cases the prognosis is graver than in the toxic cases. The symptom-complex is found, although comparatively seldom, in general paralysis. Brain tumour is sometimes a cause and occasionally the diagnosis from an alcoholic Korsakoff syndrome is difficult; strangulation, poisoning by carbonic acid gas and concussion of the brain exhaust the list of causes.

As to the mode of occurrence of the symptom-complex, it may be preceded by hallucinatory conditions and deliria, by epileptic or apoplectiform attacks, or thirdly by stuporous conditions. The prognosis depends upon the accompanying condition, but one can almost always conclude that the brain is severely damaged.

C. MACFIE CAMPBELL.



**ON HYSTERICAL INSANITY. (Ueber hysterisches Irresein.)**  
(148) RAECKE (of Frankfort), *Allg. Ztschr. f. Psych.*, Sept. 2, 1904.

RAECKE bases his remarks on a material of 168 observations; in the Frankfort Asylum cases of hysterical insanity formed 4 to 6 per cent. of all the cases. The author agrees with Binswanger that a psychosis is only to be called hysterical if it proceeds directly from elements of undoubtedly hysterical nature; he therefore first discusses the simple transitory disturbances of consciousness of the hysterical.

In hysteria, even in the absence of what is usually called mental disturbance, hallucinations are not uncommon and usually occur at night. They are usually visual, of a terrifying nature, are recognised as illusions, but may cause lively anxiety. Paranoid ideas may appear episodically, the insane jealousy of the hysterical is well known, hysterical symptoms may undergo elaboration into hypochondriacal ideas, ideas of greatness may spring from mere romancing. The emotional variations of the hysterical are important; the extreme forms are the *raptus hystericus* with its extraordinary anxiety, accompanied by motor unrest, palpitation, and occasionally leading to attempts at suicide or to deeds of violence, and the *furor* or maniacal excitement often brought on through passion, alcohol, or a hysterical convulsion. Other hysterical episodes are the various states of hazy consciousness and dream states (Dämmerzustände) with all transitions from the waking dreams, due to an unbridled imagination, to states of somnambulism and the phenomena of double personality. Hysterical stupor is another element which may occur apart from, or as an element in, hysterical insanity. The most typical mental disturbance of the hysterical is a delirium based on the recollection of a real event or of some story which has deeply impressed the imagination; the delirium is usually terrifying, but occasionally of ecstatic nature. Hysterical insanity not only proceeds from these elements, it is actually composed of a series of waking dreams, hallucinations, Dämmerzustände, emotional variations, stuporous and delirious states in every possible combination.

Although it presents such a variable picture, there are two main forms, a melancholic and a paranoid. In the former there are terrifying deliria, fits of anxiety with attempts at suicide, a succession of waves of depressed feeling which colour the ground, upon which appear paranoic episodes, maniacal outbursts, intervals of trifling gaiety. The paranoid form is of slower development; waking dreams and visions seen at night, are elaborated by the romancing imagination into a system which only has transitory mastery of the patient, but which after a long interval may reappear, usually as the result of a Dämmerzustand; while phases

of stupor and delirium, episodes of somnambulism and double personality may at any time appear. In discussing the diagnosis of hysterical insanity, Raecke lays stress on its relation to outer causes, the superficiality of the phenomena, the reaction to suggestion. It is impossible to foretell the course of the individual case, but the paranoid form seems to run a more chronic course.

C. MACFIE CAMPBELL.

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## Review

**TRATTATO DI PSICHIATRIA.** Prof. LEONARDO BIANCHI. Casa Editrice Cav. Dott v. Pasquale, Napoli, 1905.

THE first part of this work opens with a short description of the cerebral surface, followed by that of the olfactory, optic and auditory centres and paths, along with those of common sensibility and motion. These tracts are described very clearly and followed continuously from their origin to their final termination. A description of the minute anatomy of the cerebral cortex comes next, attention being paid to the work done by the Golgi and Nissl methods. In the discussion on the neuron theory, Bethe's and Apathy's work on the neurofibrils is naturally referred to, as well as that of Fragnito and Capobianco on the multicellular origin of the nerve cell. The remainder of Part 1 is taken up with the physiology of the cortex. The localisation work of Ferrier, Horsley, Luciani, Bianchi, Hitzig, etc., is fully described and ably criticised, as well as the embryological researches of Flechsig. This chapter with that on Language is splendidly treated.

Part 2 is devoted to Psychology, and is invaluable to the student of psychiatry. Professor Bianchi is to be congratulated on the clear, logical way in which he places the physio-pathology of attention, perception, memory, etc. before the reader. The authorities quoted are numerous, and the criticisms of the author justify themselves at all times by the precision of his argument.

Part 3 consists of 432 pages, and covers very thoroughly the field of Clinical Psychiatry. At the beginning the author gives his method of examination of the mental state, and then describes the various types of insanity. Every variety is fully dealt with, and not only are the clinical features minutely described, but the psychology of Part 2 is applied in this section with great advantage.

The chapters on Idiocy, Imbecility, Crime, Paranoia, Epilepsy, and General Paralysis are particularly fine, and the cases used as illustrations are given with a detail which denotes the most careful observation. The intoxication theory of Ceni on epilepsy is not quite accepted by the author, who lays great stress on the importance of a neuropathic heredity. According to him the

presence of traumatic and organic lesions, alcohol and syphilis, causing alterations in the fine structure of the brain, are important

In discussing the etiology of General Paralysis, one is struck by the unusual absence of narrowness or of bigotry in the author's consideration of the subject. The statistical work of himself and others has been carefully scrutinised and weighed. Syphilis, alcohol, bad heredity, trauma, venereal excesses, apoplexy, preceding mental maladies, nephritis, may each induce a bio-chemical (toxic) condition which lowers the nutritive energy of the nerve elements and favours the final predominance of the connective elements. The lowered resistance of the organism prepares the soil for an intoxication which may or may not be microbiotic.

One of the most interesting chapters in the book is that on "Frenosi Sensoria," a subject made specially interesting by the author's personal researches. Under this term are included all psychopathic forms beginning with hallucinations and illusions. These form the most conspicuous feature in the whole course of the disease, or disappear early, leaving symptoms such as amentia, dementia, katatonia, stupor, dementia præcox, and mental confusion. These, according to Professor Bianchi, are only peculiar phases of the malady and not individual diseases. The early hallucinatory phenomena disintegrate the personality, leaving it so, or its components may be rearranged, giving rise to katatonia or paranoid phenomena. Professor Bianchi's opinion is certainly supported by the cases cited, and one welcomes his determination to group these symptoms into one clinical entity.

The whole work of 844 pages ranks as one of the finest on the subject. Although such a wide field is covered, each part is treated in the most thorough manner and brought up to date.

Professor Bianchi gives to us the result of wide reading, years of study and work in his laboratory and clinic; this, coupled with a beautifully clear style and lucidity of expression and explanation, makes his treatise a genuine pleasure to read. Such a work is just to be expected from one equally at home in the laboratory, psychiatric clinic, or in the asylum wards, and at the same time is an expression of the splendid and advanced work which has been going on in Italy for years.

DAVID ORR.

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\* A number of references to papers on Treatment are included in the Bibliography under the individual Diseases.

# **Review**

or

# **Neurology and Psychiatry**

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## **Original Articles**

### **ON THE PRESENCE OF DIPHTHEROID BACILLI IN THE GENITO-URINARY TRACT IN CASES OF GENERAL PARALYSIS AND TABES DORSALIS.**

By W. FORD ROBERTSON, M.D., and G. DOUGLAS M'RAE,  
M.B., C.M., M.R.C.P.Ed.

IN previous papers (1), on the ground of investigations made by Dr John Jeffrey and ourselves, the hypothesis has been advanced that general paralysis is the result of a chronic toxic infection from the respiratory and alimentary tracts, permitted by general and local impairment of the defences against bacteria, and dependent upon an excessive development of various bacterial forms, but specially upon the abundant growth of a diphtheroid bacillus, which gives the disease its distinctive characters. The recorded results of our bacteriological and histological researches are in harmony with this view, and the experimental observations, carried out by Dr Shennan and one of us (2), upon rats, at least tend to support it. As is well known, the hypothesis that is accepted by the great majority of neurologists at the present day is that general paralysis is essentially syphilitic in origin, the most common explanation being that the antecedent syphilis has in some way brought about a progressive disorder of metabolism, which results in a special form of auto-intoxication. It is, however, easy to trace in recent literature a growing feeling that this view does not fully harmonise with the known facts. We have never denied that syphilis plays an important part in the etiology of general paralysis, but we have maintained there is no satisfactory evidence

of its rôle extending beyond that of weakening the general immunity.

The more recent observations that we wish to record briefly in this paper, incline us to attach a more specific importance to the diphtheroid bacillus than we have hitherto felt warranted in doing. Indeed, they raise the question whether or not general paralysis and tabes dorsalis are dependent upon a special infection, commonly venereal, which is neither that of syphilis nor of any other as yet recognised venereal disease.

Continuing our investigations into the distribution of diphtheroid bacilli in general paralytics, we have sought to ascertain if these organisms are commonly present in the genito-urinary tract. A fact of great significance that has been elicited in the course of this investigation is that all the female general paralytics examined were suffering from chronic leucorrhœa. In these patients the discharge has always been of a creamy, purulent character. This morbid condition may escape the notice of the nurse, but can at once be detected by the use of the speculum. The amount of the discharge varies very considerably from time to time in the same individual. This symptom has been observed in all of the last thirty-six female general paralytics admitted to the Royal Edinburgh Asylum.

We have made direct film preparations and cultures from the vaginal discharge of fourteen female general paralytics, and from the surface of the urethra of twenty-two male general paralytics. In every case we have found diphtheroid bacilli to be present. They were generally visible in large numbers in the direct films, and we have rarely had any difficulty in finding colonies containing the same organism upon the byno-hæmoglobin-agar<sup>1</sup> medium. In two male and in two female cases the first growths were pure. In all the others they were mixed. A diplococcus was the most frequent associate of the diphtheroid bacilli; streptococci, staphylococci, and the bacillus coli were also common.

Lest there should be any misunderstanding as to what is

<sup>1</sup> We now recommend that this culture medium be prepared as follows:—To 500 c.c. of water, placed in a sterilized litre flask, add 10 grammes of agar. Boil until the agar is dissolved. Then add 25 c.c. of byno-hæmoglobin (Allen & Hanbury's). Sterilize in the autoclave for half an hour at 110° C. Filter through a linen cloth, or plug of cotton wool. Add 30 c.c. of glycerin. Fill into tubes. Sterilize in autoclave for one hour at 110° C. Slope the tubes until the medium has solidified. Incubate for twenty-four to forty-eight hours before using.

implied by the term "diphtheroid bacillus," we would state that we mean simply a bacillus which has the general cultural features and the staining reactions to Neisser's method which characterise the Klebs-Löffler bacillus.

The application of similar methods to the subjects of other forms of insanity has revealed the fact that diphtheroid bacilli are very commonly present in the genito-urinary tract in cases in which there is no clinical ground for suspecting that the patient is suffering from general paralysis. Three out of six females were positive and six out of seven males. The bacilli were as a rule notably few in number. The possibility of some of these positive cases being incipient general paralytics cannot be excluded.

On first view the results of these control observations might seem to dispose entirely of the contention that importance is to be attached to the presence of diphtheroid bacilli in cases of general paralysis. A little further examination of the question should, however, make it apparent that it cannot be so easily dismissed. A like method of reasoning would compel us to refuse to attach any importance to the presence of the Klebs-Löffler bacillus in cases of acute diphtheria, for bacilli morphologically identical are very common, in the throat and elsewhere, in other morbid conditions and even in health. In these instances they may even be virulent in character.

With regard to the question of the frequency of the occurrence of diphtheroid bacilli in the genito-urinary tract of the female, it is of interest to note that Foulerton and Victor Bonney (3) in a recent paper state that they found an organism of this kind in the uterus and vagina in only two cases of puerperal infection out of fifty-four examined.

It seems to us that the known facts regarding "diphtheroid bacilli" warrant the following general conclusions. Bacilli of this morphological character form a very wide genus, some of the separate species of which have already been distinguished on the ground of cultural and pathogenic characters. Some forms are harmless saprophytes which are commonly present in the alimentary and upper respiratory tracts, less frequently in the vagina and male urethra. Many of the forms are highly polymorphic, and the virulence of some is liable to be raised by growth under conditions as yet little understood. Representative of an extreme

of virulence in one direction, there is the bacillus which has been demonstrated to be the essential pathogenic agent in acute diphtheria. This organism is virulent for guinea-pigs and innocuous to rats. Having an extreme of virulence in another direction, there is a form which has been isolated from cases of general paralysis and which has been proved by our investigations, already referred to, to be virulent for rats and innocuous to guinea-pigs.

It still remains to be proved whether or not this last form is the essential pathogenic agent in general paralysis. The question can probably be determined definitely only by experiment upon animals. The investigation of this nature, above alluded to, though not sufficiently extensive to be decisive, has yielded evidence in favour of a positive conclusion.

Dr Lewis C. Bruce has kindly allowed us to refer to an experimental observation that he has recently made upon a goat. From time to time in the course of several months the animal was injected subcutaneously with cultures of a diphtheroid bacillus, isolated from a case of general paralysis, for the purpose of obtaining an immune serum for therapeutic use. The cultures used were derived from the same case as those that were chiefly employed in the experimental investigation upon the rats. After a time the animal developed signs of severe alimentary disturbance. It had been known to lick the spots at which the injections were made. It became tottering in its gait, and about six months from the time when the last subcutaneous injection had been made, it had a seizure closely resembling the congestive attack of a general paralytic. It rallied to some extent, but died a few days later. A culture was made from the oesophagus after death, and a growth of a diphtheroid bacillus was readily obtained. Dr Bruce sent the brain to us for examination, and we have ascertained that it shows changes resembling those found in general paralysis.

Some other recent observations of our own also seem to us to favour our hypothesis, and especially to throw new light upon the relation of a diphtheroid bacillus to tabes.

We have at present under observation a case of advanced locomotor ataxia which has developed the signs and symptoms of general paralysis. Having found a diphtheroid bacillus in very large numbers in direct film preparations, as well as in a culture,

from the urethra, it occurred to us that it would be important to ascertain if the same organism was present in the bladder. Two cultures were made from the flowing urine towards the end of micturition, when it might be presumed any organisms lying free in the urethra would have been cleared away. The tubes, after twenty-four hours' incubation, each yielded a few large colonies of a coccus and innumerable small colonies of a diphtheroid bacillus. This patient has no symptoms of bladder irritation.

In a similar case in a woman we have also been able to determine by withdrawing the urine by means of a sterilized glass tube and centrifuging, that a diphtheroid bacillus is present in the bladder in very large numbers. It is associated with another bacillus.

From another case of tabetic general paralysis in a male we obtained a small quantity of urine towards the end of free micturition. This was centrifuged, and a film made from the deposit showed, when stained by Neisser's method, fields packed exclusively with diphtheroid bacilli.

Urine from another case of tabetic general paralysis in a woman was similarly treated, and showed the presence of abundant organisms, which again seemed to be chiefly diphtheroid bacilli.

In five cases of general paralysis and two controls, a platinum loopful of urine was taken from the stream during micturition and stroked upon byno-hæmoglobin-agar. In each instance the medium remained sterile after forty-eight hours' incubation. In two of these cases it had previously been ascertained that the epithelium of the anterior part of the urethra contained abundant organisms of various kinds, and therefore it seems to us we are justified in concluding that in the tabetic cases the organisms were derived from the bladder.

Orr and Rows (1) have recently concluded, from the results of a very careful investigation, that the initial tabetic lesions in the spinal cord are caused "by some noxious agent which circulates in the lymph and acts on the nerves at the point where they lose their neurilemma sheath." If, as would appear from the results obtained in the four cases just cited, a diphtheroid bacillus is commonly present in the bladder in *tabes dorsalis*, and if these bacilli have the virulence of those that proved fatal to several rats, the cause of the toxic lesions in the



cord and posterior roots is not far to seek. It is probable that in these cases the diphtheroid bacilli extend much further up in the urinary tract than the bladder. In a case of advanced general paralysis in a woman, recently examined post-mortem, we have found a bacillus of this kind to be present in great numbers on the surface of the bladder, ureter and pelvis of the kidney.

In two cases of general paralysis we have lately found, after death, a localised purulent cerebral meningitis. We made cultures from the purulent material, and in both instances obtained growths of a diphtheroid bacillus. In one of the cases this organism was associated with a colon bacillus, and in the other with the pneumococcus. Including these two cases, we have now ascertained by cultural methods the presence of a diphtheroid bacillus in the brain of general paralytics in seven cases out of twenty examined.

During the last two years we have from time to time endeavoured to observe if these diphtheroid bacilli display any special reaction towards the blood of the general paralytic. We have been unable to get any decisive results by agglutination methods, but, somewhat recently, with the aid of a new technic, we have in some cases obtained a reaction of a positive kind, which seems not only to afford evidence that the organism is exerting a pathogenic action, but also to provide a method of serum diagnosis. We hope to be able to deal with this subject in an early paper.

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1. *Review of Neurology and Psychiatry*, April, May, and July 1903 ; *Brit. Med. Journ.*, October 24, 1903.
2. *Review of Neurology and Psychiatry*, April 1903.
3. *The Lancet*, April 8 and 15, 1905.
4. Orr and Rows, *Brain*, Winter 1904.

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### SOME ASPECTS OF ALCOHOLISM.

By A. HILL BUCHAN, M.A., M.B., M.R.C.P.E.

(Continued from p. 182.)

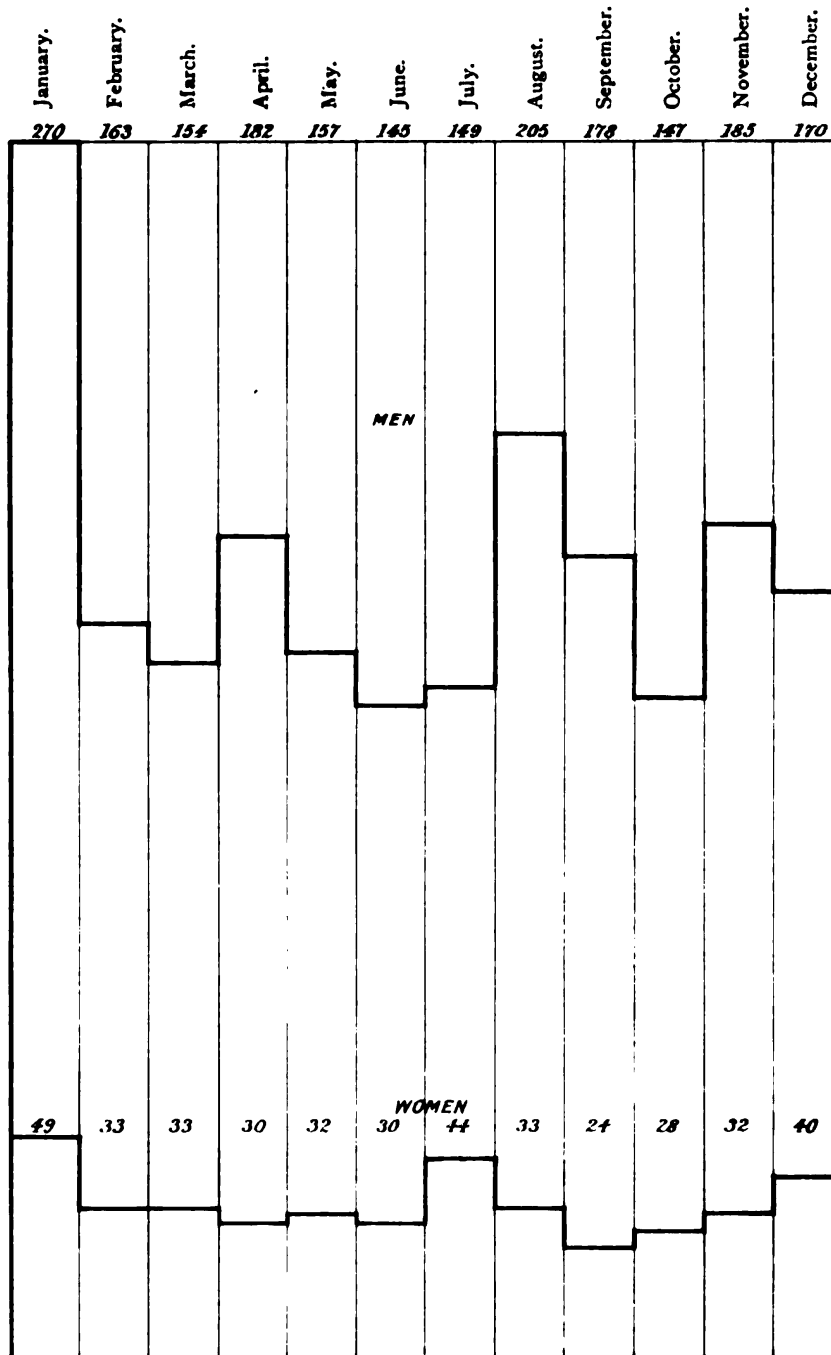
#### SEASONAL INCIDENCE.

THE question of whether meteorological conditions have any influence, direct or indirect, on the occurrence or severity

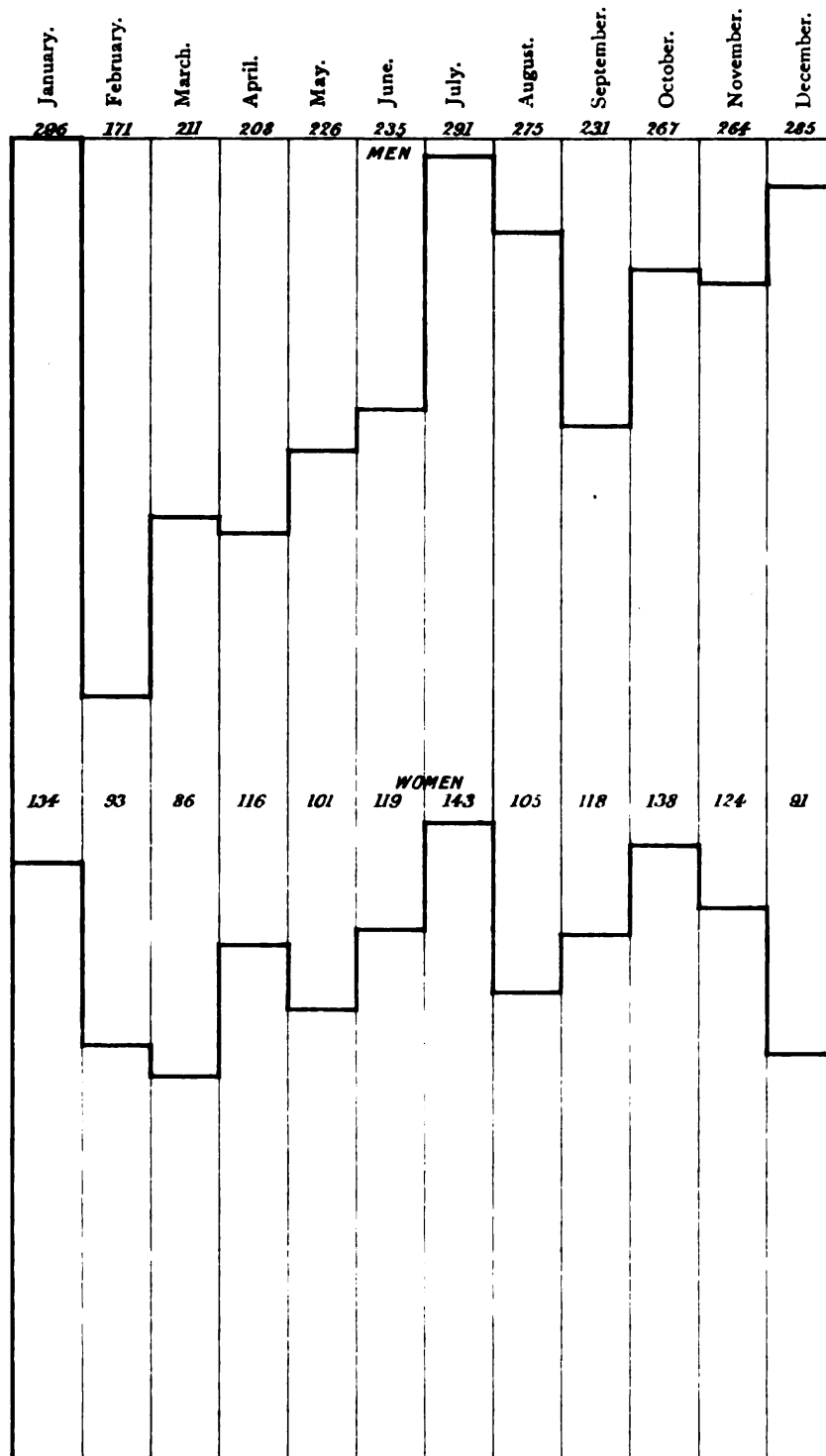


# CASES OF DELIRIUM TREMENS

(Extending over 45 Years).



OTHER CASES OF ALCOHOLISM  
(Extending over 45 Years).





of the effects of alcoholism, is a complicated one. The times of holidays, especially New Year and "Trades" holidays, the dates when wages or pensions are paid, variations in the state of trade, etc.—these and other factors obviously play a rôle so important as to render conclusions as to the influence of season very uncertain.

Charts were made of the seasonal incidence of the series of cases which have been under review, but it was felt that 5 years was too short a period. Accordingly an additional examination was made of the record of admissions to the ward preceding this time, 45 years in all being examined. I am indebted to Dr Alexander Bruce for the accompanying charts of seasonal incidence for that period of 45 years. The cases have been arranged according to sex. The first shows all the cases of D.T.; the second, all admissions for any other effect of alcoholism.

The large number of cases in January, both of D.T. and other forms of alcoholism, is noticeable.

The summer maximum of D.T. cases for men occurs a month later than that of other forms of alcoholism, almost suggesting that such a period of drinking is needed for the development of D.T. in the majority of individuals. It will also be borne in mind as to the winter maxima that the drinking bout usually commences about the beginning of January or end of December.

In conclusion, I wish again to express my heartiest thanks to Dr Alexander Bruce for so kindly allowing me to make use of the records of his cases for the preceding papers.

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### **A CASE OF ACUTE ASCENDING PARALYSIS, WITH AUTOPSY.**

By EDWIN BRAMWELL, M.B., F.R.C.P.E., M.R.C.P. Lond.

THIS is a clinical and pathological report of a case of acute ascending paralysis which presented somewhat unusual features.

#### **CLINICAL RECORD.**

ON December 3, 1904, I was asked by her medical attendant to see a married woman, forty-six years of age, who for a fortnight had been suffering from *severe pain in the back*. The patient stated that she had been perfectly well until two weeks previously, when the pain above referred to began to trouble her. She said she felt the pain over the small of the back, that at

first it was not severe though never absent, but that it had been very intense during the past week. The severity of the pain may be gauged from the fact that the patient, naturally an active, energetic woman, had remained in bed during the previous ten days, that she had had little sleep, and had required to have morphia injections. At the time when the pain first appeared she complained of some *numbness in the arms* which had been present more or less up to the time of my visit; she said she felt as if she were unable to use her hands properly. On December 1, her doctor noticed that the *right side of her face was paralysed*; this developed apparently without any subjective sensations. For twenty-four hours previous to my examination, the pain in the back had been decidedly less severe, but a *gnawing, restless feeling in the legs* had prevented her from sleeping. During the night of December 2, she was said to have had several attacks of shaking in the arms and legs, but the account of these attacks which we received was indefinite. There had been no headache or vomiting, and the temperature had been normal throughout.

The patient had, previous to this illness, enjoyed very good health. Her doctor, who had attended her for fifteen years, was of opinion that alcoholic abuse might be definitely excluded, and from the patient's husband we subsequently ascertained that she, though not a teetotaler, rarely took any alcoholic drink. She had never had rheumatism, and (the doctor stated) there had never been any symptoms suggestive of syphilis.

*Physical examination on December 3, 1904.*—The patient was a well-developed, well-nourished woman, and was not anæmic. Mentally there appeared to be nothing abnormal, she answered questions intelligently and displayed no undue emotion. Speech, articulation, and phonation were unaffected. The optic discs were normal. She heard my watch ticking six inches from either ear. She had never had any ear discharge. The pupils were equal and reacted well. The ocular movements were perfect, and there was no nystagmus. The masseters and temporals contracted well, and there was no facial anæsthesia. There was complete *right-sided facial palsy of peripheral type*, she could not close the right eye nor wrinkle the forehead. The palate moved well and symmetrically. The tongue was protruded straight (slight apparent deviation was due to the unilateral facial weakness). Movements of hands and arms were satisfactory on both sides. She carried out the finger-nose test perfectly accurately with either hand (*i.e.* there was no ataxy). She complained of being unable to use her hands properly, but she buttoned her night-dress quite well. No anæsthesia, analgesia or thermanalgesia was detected on the hands, arms or trunk, and there was no defect of the stereognostic sense in either hand. The movements of the lower limbs were also satisfactory, and there was no rigidity. She raised both feet off the bed perfectly steadily, and touched a finger held a foot above the bed. She walked somewhat unsteadily, but this might well have been accounted for by the circumstance that she had been in bed for ten days. There was no Rombergism. I was unable to detect any anæsthesia or analgesia of the lower limbs. The knee-jerks were present, equal, and about normal in activity. There was no ankle-clonus. The plantar reflexes were absent. She had had no difficulty in making water. There was no vertebral irregularity or prominence and no tenderness on pressure over the spine. The back muscles were not tender, even on deep pressure. The heart sounds were pure, the second sound at the base being a little accentuated. There was no outward displacement of the apex, and the radial artery was not thickened. The lung resonance posteriorly extended down to its normal limits. The urine contained a few pus cells.

*Summary.*—To sum up the case at this stage:—A perfectly healthy woman develops, without apparent cause, a very severe

pain in the back at the junction of the middle and lower thirds of the dorsal region. The onset of the pain was associated with temporary numbness in the arms. About a fortnight later a right facial palsy appears, the pain in the back was now disappearing, but she complained of subjective sensations in the legs. Upon examination, no objective sign of disease was detected with the exception of a right-sided peripheral facial palsy.

On December 8, when I again saw the patient, the clinical picture of the case had considerably changed. There was then marked *general weakness of the lower limbs*, especially of the right, which she was unable to draw up in bed against slight resistance. There was, however, no special weakness of the dorsiflexors of the feet. There had been no recurrence of the back pain, but the patient complained of a constant restless feeling in the legs. The feet, she said, felt cold and numb. The *knee-jerks could not be elicited*, even with reinforcement. The *plantar reflexes were not obtained*, the soles of the feet were not hyperæsthetic. The muscles of the *calves were distinctly hyperalgetic* on pressure, but the hyperalgesia was not excessive. The *grasp was perhaps a little feeble, rather more so on the right than on the left side*. With eyes closed she missed the nose by  $1\frac{1}{2}$  inch or so with the right hand, with the left hand the movement was satisfactorily carried out. There was now *distinct weakness of the face on the left side*, though the paralysis was not nearly so marked as on the right. There was no difficulty in swallowing. The voice was unaffected. The right pupil was a shade larger than the left, but both reacted quite well. There was no ptosis or strabismus. The ocular movements were quite satisfactory. The patient could see to read small print. There was no weakness of the diaphragm or intercostals. There was no cough. The patient looked ill, and movement appeared to be very exhausting. The temperature was normal. The pulse-rate was 80, and the beats were regular in time and force. The spleen and liver were not palpable. The heart sounds were closed; the first sound was, however, reduplicated at the apex, and perhaps a little feeble. The apex-beat made out by percussion was in the fifth space internal to the nipple, and no impairment of the percussion note to the right of the sternum was detected.

The patient now stated that she had had a *sore throat three weeks or so before the present illness began*. The sore throat had lasted for a week, during which time she had considerable pain on swallowing, and did not feel at all well. Both the patient and her husband thought that she had acquired it from her son, who was suffering from a sore throat which she was swabbing at the time. She said she was not subject to sore throats. No one else in the house was ill, and we were unable to elicit a history of any other symptoms at the time of the sore throat which might have given a clue as to its nature. Upon ascertaining this history we made a careful examination of the throat, but excepting for a little congestion of the fauces and pharynx there was nothing abnormal present. Swabs were sent to the Royal College of Physicians Laboratory, and from inoculations a growth of staphylococci was obtained.

*Summary.*—From the account of the patient's condition on December 8, it will be seen that there had been a considerable development of symptoms between this date and the occasion of my first visit five days previously. There was now a bilateral



facial palsy ; marked weakness of the lower extremities, affecting especially the right, but not picking out any special muscle group ; slight weakness in the hands, more particularly the right ; subjective sensations of cold and numbness in the feet, and some hyperalgesia of the calf muscles ; absence of the knee and ankle-jerks. In addition, we obtained the definite information that the patient had suffered from a sore throat some three weeks before the onset of her illness. As to the nature of the sore throat we had no evidence.

On December 14, as the patient's condition was becoming worse, she was admitted to the Edinburgh Royal Infirmary, under the care of Dr Byrom Bramwell. I have to thank Dr Davidson, the house-physician, for his notes of the case, from which the following extracts are taken :—

*State on admission.*—There is almost complete paralysis of both legs. There is marked weakness in both arms, but all movements can be performed. There is complete paralysis of the right side of the face, and marked paresis of the left side. The tongue is protruded straight, but is dry, slightly furred, and brown. The voice is very low and husky. There is no affection of the eye muscles.

The organic reflexes were unaffected till 15th, when, having passed no water, a catheter was used to draw it off. The other organic reflexes were unaffected. The epigastric, abdominal, and plantar reflexes could not be elicited. The ankle and knee-jerks were absent. The supinator and biceps-jerks were active and equal, and the triceps-jerks were elicited with some difficulty.

The muscles are tender if pinched at all hard. The patient has never had any diplopia.

There appears to be no alteration in sensibility. She has no pain. The pupils react to light and accommodation, are equal and regular, measuring 4 mm. in diameter. She hears and understands what is said to her quite well. There is some mental impairment, but no delusions. The pulse-rate is 90 ; it is rather irregular in force and weak ; both the expansion and pressure are poor. The heart is not enlarged, and the sounds are pure. The specific gravity of the urine is 1025, the reaction acid, and there is a trace of albumen. The temperature is 98°.

*On the morning of December 16.*—The pulse is rather stronger. The paralysis of the left side of the face seems more marked, and the left arm is also weaker. There is no difficulty in swallowing and no nasal regurgitation. Expansion of chest defective. There is some weakness of both intercostals and diaphragm. Marked alternation of thoracic and abdominal expansion (E. B.). She has a feeble cough, and appears to be unable to clear her throat. Some dulness and crepitations at both bases.

The right side of the face reacts very feebly to a strong faradic current, and A.C.C. is almost equal to K.C.C. The faradic excitability of the left side of the face is somewhat reduced. The muscles of the lower extremities react to faradism, but the contraction is a poor one.

*December 17.*—Patient complains of a tight feeling beginning in left breast and passing down left side to above pubes, and up right side to right breast—a feeling as if a tight rope has been bound round her. A blood count shows no leucocytosis.

*December 18.*—Patient has considerable difficulty in breathing; there is a good deal of rattling in the throat, which she appears to be unable to clear. Pulse 110; respirations 30; temperature 98°. Quite delirious. No difficulty in swallowing, and no regurgitation or choking. Her voice is sensibly weaker. Accessory muscles of respiration are acting much. Heart sounds faint. No murmurs. Urine's specific gravity 1022, acid, no albumen. No perceptible change in the state of the nervous system.

*At 7 p.m.* the same evening, breathing laboured; respirations 34; pulse 115, somewhat irregular, pressure low; temperature 98°.

*At 11 p.m.*, respirations 44; pulse 120; temperature 98.2°; chest moving as a whole; accessory muscles in neck acting very powerfully; abdomen not moving nearly so powerfully as before.

Patient died at 12.30 p.m.

#### POST-MORTEM EXAMINATION.

An autopsy was made eleven hours after death by Dr Stuart M'Donald whom I have to thank for the following notes:—

There were no abnormal external appearances beyond some dark bruise-like areas over the right shoulder, and especially on the back, on both sides of the spine almost in its entire length.

*Heart.*—Nothing abnormal on surface. No pericarditis. No endocarditis. Some chronic degeneration in the anterior cusp of mitral valve. A few petechial hæmorrhages below endocardium of the left ventricle, just below the aortic cusps. Heart muscle is soft and flabby, but shows no distinct fatty change; both orifices are slightly dilated; there is some very slight atheroma of the aorta, but none of the coronary arteries.

*Lungs.*—Some few chronic adhesions on both sides; no fluid. The right lung shows well marked hypostatic congestion and some distinct consolidation in the lower lobe, one of the consolidated areas measuring fully an inch and a half in diameter. The upper lobe is congested and shows small areas of hæmorrhagic infiltration, but no distinct consolidation. The left lung shows emphysema and hypostatic congestion as on the other side; in the lower lobe some small areas of broncho-pneumonic consolidation are present. No tuberculosis in either lung.

*Abdomen.*—The mesenteric glands show some slight enlargement, one is calcareous evidently from old tuberculosis.

*Stomach and small intestine* dilated; nothing else abnormal beyond some chronic adhesions around the spleen and ascending colon.

*Spleen.*—Slightly enlarged and soft. Malpighian bodies are unduly prominent.

*Liver.*—Fatty.

*Gall-bladder.*—Shows nothing special.

*Kidneys.*—The left kidney is somewhat horseshoe-shaped, and lies at the level of the pelvic brim, to which it is adherent. The left suprarenal lies in its normal situation, i.e. it has not been displaced downwards with the kidney. The cortex of both kidneys is slightly swollen, and there is slight chronic interstitial change with some acute change superadded, the latter probably from the lung condition.

*The bruise-like areas on the back* proved to be hæmorrhagic, and there were many similar areas situated deeply actually in the muscles. There were no purulent deposits in relation to the hæmorrhages or elsewhere. The hæmorrhages suggested some toxic process.

*Brain and cord.*—The cerebral cortex was somewhat congested and cedematous. The grey matter of both the brain and cord were congested. There appeared to be some excess of cerebro-spinal fluid; there was no evidence of meningitis.

## SUMMARY OF CASE.

### CLINICAL.

Patient, a married woman, aged 46. Previous health excellent. A "sore throat" of undetermined nature. Three weeks afterwards very severe pain in back, which persisted for a fortnight, also some numbness of hands. Ten days after "back pain" appeared, *right-sided* peripheral facial palsy developed. Two days later uncomfortable sensations in legs. At this time nothing objective detected, excepting the facial palsy above mentioned.

Patient seen again four days later. Marked weakness of legs, slight weakness of arms, and left side of face now parietic. Knee and ankle-jerks not elicited. No anæsthesia detected. No bulbar or ocular symptoms.

Subsequently paralysis of legs became complete, and weakness of arms more pronounced. Respiratory muscles affected. No bulbar symptoms. Pneumonia (temperature normal throughout). Death. Alcohol could be excluded. No evidence of influenza or diphtheria. (Total duration of illness about four weeks.)

### PATHOLOGICAL.

#### 1. POST-MORTEM (*within twelve hours of death*).

*Brain and spinal cord*.—Grey matter congested.

*Lungs*.—Hypostatic congestion and broncho-pneumonia. Some few adhesions on both sides.

*Heart*.—Muscle soft and flabby. A few subendocardial petechial hæmorrhages.

*Spleen*.—Slightly enlarged and soft.

*Kidneys*.—Slight swelling of cortex and chronic interstitial change.

Mesenteric glands slightly enlarged.

Hæmorrhages of considerable size into muscles of back.

#### 2. HISTOLOGICAL EXAMINATION OF NERVOUS SYSTEM.

Parts examined (*vide* diagram on opposite side).

Methods used:—Busch, Nissl, Weigert-Pal, Heller, van Gieson, Haem and Eosin.

Great vascular engorgement, numerous capillary hæmorrhages, all apparently of recent origin, in cord, particularly into grey matter.

No obvious changes in vessels.

No definite pathological changes in nerve fibres of cord.

*Nerve cells*.—Cortex normal.

Pronounced changes in both seventh nuclei, especially right.

Marked changes in cells of lower part of cord, becoming less evident at higher levels.

Intervertebral ganglion (S. 1), slight changes (?).

*Peripheral nerves*.—Anterior and posterior roots (S. 1) showed no change.

Degeneration with Busch's method in right facial and external popliteal nerve.

In ulna and phrenic nerves nothing abnormal detected.

*Muscle*.—Diaphragm showed no change.

Tibialis anticus presented an acute degeneration.

#### 3. BACTERIOLOGICAL EXAMINATION.

*Ante-mortem*.—Swab from throat—staphylococci.

*Post-mortem*.—Cerebro-spinal fluid in glucose broth and blood agar negative.

Blood from right ventricle in glucose broth and blood agar negative.

Splenic puncture in glucose broth negative.

Section of spinal cord (L. 1) and muscles of back into which hæmorrhage had occurred (Thionin blue, Grams, Z. Neelsen, and a modified Benda method) negative.

Epidual tissue, teased and stained with methylene blue, negative.

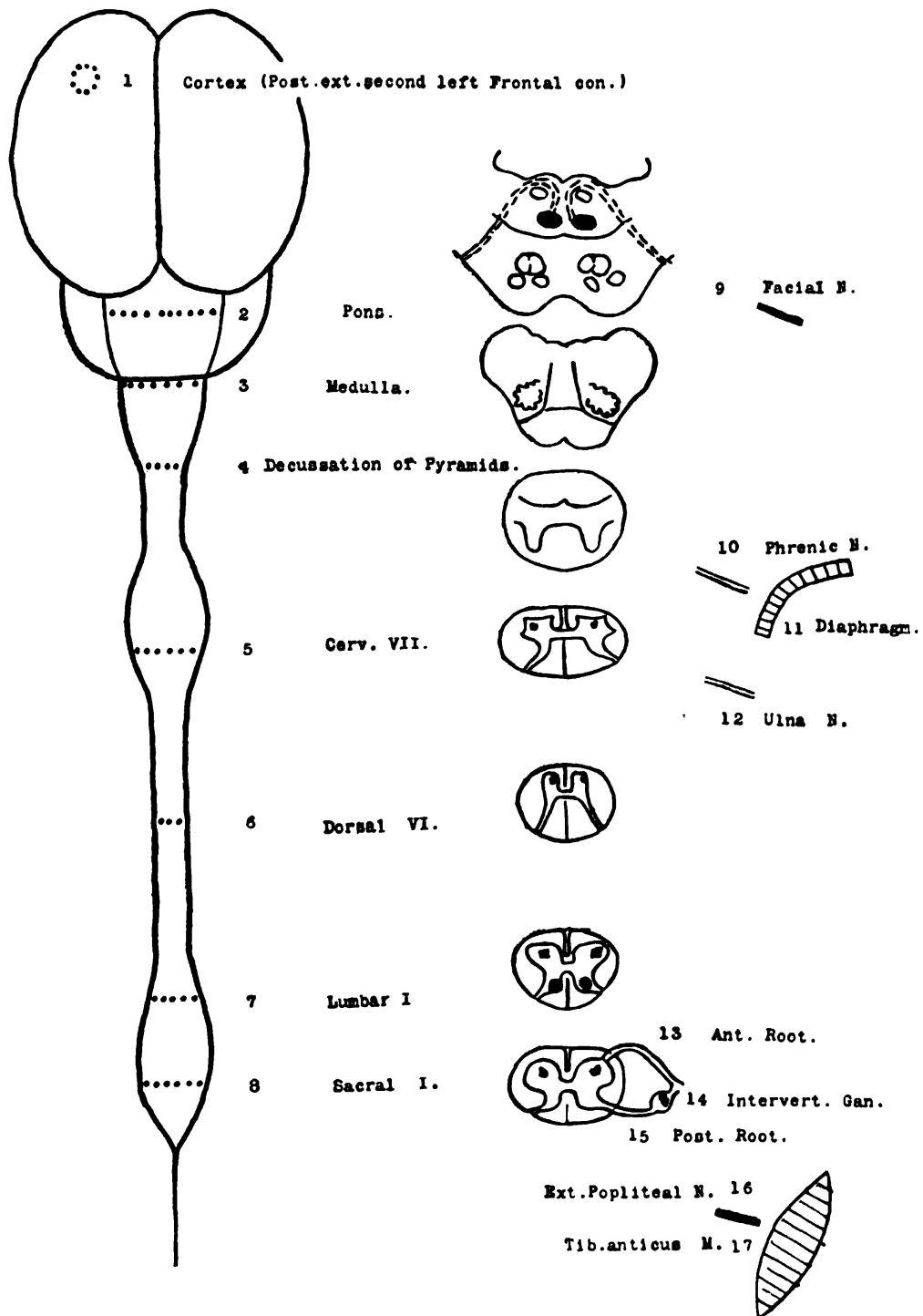


Diagram to show the parts of the nervous system examined. Structures in which pathological changes were met with are marked in red.





FIG. 1.



FIG. 3.



FIG. 5.

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*Bacteriological Examination.*—The following inoculations were made :—

- (a) A loopful of fluid from below the dura in glucose broth.
  - (b) A loopful of cerebro-spinal fluid in glucose broth, also on blood-agar.
  - (c) The surface of the right ventricle having been sterilised, about 1 c.c. of blood was withdrawn with a sterile syringe and divided between a glucose broth tube and an agar tube.
  - (d) A glucose broth tube was inoculated from the spleen.
- All of these tubes were sterile after ten days.

### HISTOLOGICAL EXAMINATION (by E. B).

The following tissues were microscopically examined :—(a) spinal cord, (b) medulla, (c) pons, (d) a piece of cortex from posterior extremity of second left frontal convolution, portions of (e) right facial nerve, (f) left external popliteal nerve, (g) right phrenic nerve, (h) left ulna nerve, (i) the first sacral intervertebral ganglion from the right side, (j) the first sacral anterior and posterior nerve roots, (k) diaphragm, (l) left tibialis anticus muscle (m) a piece of muscle into which hæmorrhage had occurred.

*Method.*—All the above-mentioned tissues were placed for two days in 10 per cent. formalin. After two days in formalin some of the tissues were removed and placed in Busch's fluid and some in Weigert's chrome alum. The tissues were cut in celloidin, some having been hardened in formalin only, some in chrome alum, and some prepared by the method of Busch. The staining methods used were Busch, Weigert-Pal, Ford Robertson's modification of Heller's method, van Gieson, hæmatoxylin and eosin and a toluidin blue modification of Nissl's method.

### Results.

*Cerebral cortex.*—Nothing abnormal detected beyond engorgement of the blood-vessels. No hæmorrhages seen. The cells in particular show no pathological changes.

*Pons.*—Busch and Heller methods negative. The tissue prepared by Busch's method did not unfortunately include the facial nerve in its intra-medullary course. With the toluidin blue method very pronounced pathological changes are seen in the cells of both seventh nuclei. These changes are much more advanced on the right than on the left side. In the seventh nucleus on the side on which the changes are particularly conspicuous there is scarcely a single normal cell to be seen. The majority of the cells stain poorly and show advanced chromatolytic changes; many of them are swollen; in some a large, clear, rounded area occupies the greater part of the interior of the cell, the nucleus lying between this and the periphery. A considerable amount of yellow pigment is seen in many of the cells in which the degenerated changes are most marked. Many of the cells have an excentric nucleus, which in a few instances produces a distinct bulging of the cell wall next to which it lies; not more than three or four cells in each section can be considered as normal. In the opposite seventh nucleus the changes are not nearly so pronounced although perfectly definite. The great majority of the cells show well marked chromatolysis, however, while here and there cells may be seen which show the more advanced changes above described.

The cells of the sixth nucleus and those of the nuclei pontis which are seen in sections from this level present a healthy appearance.

*Medulla (through the centre of the olive).*—Sections prepared by the method of Busch show nothing pathological. The cells of the tenth and twelfth nuclei and the nucleus ambiguus showed no changes which could be considered pathological.



*Medulla (immediately above pyramidal decussation).*—Nothing abnormal detected.

*Spinal cord.*—The following segments were examined:—Cervical VII., Dorsal VI., Lumbar I., and Sacral I.

The spinal meninges, save that their blood-vessels were all engorged with blood, showed nothing to attract attention. This vascular engorgement was seen all through the cord at all the levels examined, and in almost every section small capillary hæmorrhages were met with, more especially, but not exclusively, in the grey matter. These hæmorrhages were not more numerous in the region of the anterior horn than elsewhere; they all appeared to be of quite recent origin. Accumulations of round cells and signs of inflammatory reaction were nowhere to be seen. No appearances indicative of softening were detected.

In the sections prepared by the modified Weigert-Pal and Busch's methods, no evidence of degenerated fibres was seen. It is true that in the Busch preparations there were a certain number of very fine black granules, but considering the circumstance that they were uniformly distributed through the cross section at all the levels examined, the probabilities are that they were not of pathological significance.

With regard to the *nerve cells of the cord*. At the level of the *seventh cervical segment* some of the anterior horn cells showed a slight degree of chromatolysis, but the appearances were not sufficiently pronounced in my opinion to justify one in considering them as undoubtedly pathological.

The cells at the level of the *sixth dorsal segment* presented, for the most part, a normal appearance, though a few showed some degree of chromatolysis, and here and there a cell which appeared somewhat swollen was met with.

The cells of the *first lumbar segment* showed, on the contrary, well-marked pathological changes. This was more especially the case in the cells of Clarke's column. The great majority of the cells of Clarke's column showed a pronounced degree of chromatolysis; many of them were very distinctly swollen, and in many the interior of the cells were occupied by a large clear space staining homogeneously. In most of the cells the nucleus was displaced; in some it lay against the cell wall. Not more than one or two cells in Clarke's group in each section showed a comparatively normal appearance.

Some of the anterior horn cells at this level were undoubtedly abnormal. The majority showed some degree of chromatolysis. Here and there a swollen cell was seen, and in a few there was an excentric nucleus. The degree of cell degeneration was not, however, so severe as in the case of Clarke's column.

At the level of the *first sacral segment* the pathological changes in the anterior horn cells were more pronounced than those above described, not more than one or two cells in each anterior horn presenting a normal appearance. Almost all the cells were swollen, and showed a marked degree of chromatolysis. In some there was a considerable amount of bright yellow pigment. In many of the cells the nucleus was excentric, in a few instances lying against the cell wall.

The degenerated nerve cells were scattered through the anterior horn, none of the cell groups in this situation escaping.

*Sections of the first sacral anterior and posterior nerve roots* treated by Busch's method and van Gieson presented healthy appearances.

The *first sacral intervertebral ganglion* was examined. In Busch preparations no degeneration of the fibres entering and leaving the ganglion was detected. Some of the cells stained by Nissl's method showed a degree of chromatolysis, and in some there was a large quantity of golden brown pigment, but the appearances were not sufficiently marked to enable me to say that they were undoubtedly pathological.

In the *phrenic and ulna nerves* no abnormal changes were detected. The

*right facial nerve*, on the contrary, showed by Busch's method a well-marked degeneration. By the same process similar changes were found in the external popliteal nerve although, as seen in the photograph, only a small proportion of the fibres were affected.

*The portion of diaphragm muscle* which was examined presented a healthy appearance.

*Sections of the tibialis anticus muscle* prepared by Busch's method showed a well-marked fatty degeneration in many of its fibres, about one fibre in six being involved.

*A portion of muscle from the back* into which the hæmorrhage already referred to had occurred was examined, but no evidence of inflammatory reaction obtained. Sections from this region and from the cord were stained by Mr Richard Muir for bacteria with thionin blue, Grams, Z. Neelsen, and a modified Benda method with a negative result.

Teased preparations of the epidural areolar tissue were stained by Löffler's methylene blue and examined for organisms. The result, however, was negative.

#### REMARKS.

The clinical picture presented by this case on the occasion of my first visit was to me extremely puzzling. From the subsequent development of increasing loss of power of the legs, and the involvement at a later date of the arms and respiratory muscles, the case may with certainty be designated an acute ascending paralysis. The paralysis appeared to be still progressing at the time the pneumonia, which no doubt was the direct cause of death, appeared. Had it not been for the occurrence of the pulmonary complication, it is possible that there might have been still further upward extension with involvement of the bulbar musculature.

I have avoided using the term Landry's paralysis in reference to this case, since some observers prefer to reserve this nomenclature for the exact "symptom complex" described by Landry, excluding thereby a large group of cases which present additional symptoms and variations and yet are, in many instances at least, probably dependent upon a very similar or identical pathology.

The results of the post-mortem and histological examination in this case are extremely suggestive of a toxic process acting upon the lower neurons. Thus the great engorgement of the vascular system of the cord; the numerous capillary hæmorrhages especially in the grey matter (though it seems to me difficult to exclude with certainty the mode of death as the cause of the latter, since they all appeared to be of quite recent origin); the pronounced cell changes in the lower part of the cord be-

coming more indefinite in the dorsal and cervical regions; the alterations in the external popliteal nerve and tibialis anticus muscle afford evidence which we are accustomed to associate with a toxic agent. No doubt the absence of definite changes in the cells of the upper part of the cord and of detectable alterations in the ulna and phrenic nerves, etc., is to be explained by the circumstance of their later involvement at a period before death too recent to permit of the detection of pathological alterations by the available methods of examination.

Unfortunately no evidence as to the nature of the toxic process in this case is forthcoming. It seems to me in high degree probable that the "sore throat" from which the patient suffered a few weeks previous to the onset of her illness was the source of the infection. In this relation it is perhaps of interest to note that the paralysis first appeared in the face; this may, however, have been a matter of pure coincidence. No clinical data were obtained which threw any light on the nature of the throat infection. From swabs taken from the throat a culture of staphylococci was obtained, a circumstance to which probably no importance can be attached. The inoculations made at the post-mortem were all sterile, the histological examination of the tissues for bacteria negative. In this connection I may mention that Farquhar Buzzard, in his recent valuable contribution to the pathology of Landry's paralysis,<sup>1</sup> refers to twenty-five cases in which, as in our case, bacteria were looked for and not found either by cultivations or by staining tissues or by both methods; he points out, however, that changes similar to those met with in the present case have been shown experimentally by numerous investigators to be produced by microbic toxins in the absence of the microbes themselves. He found thirteen cases of Landry's paralysis in which organisms had been demonstrated: in four of these a diplococcus was present.

Buzzard in the case which he reports found a micrococcus, which stained with Löffler's methylene blue, in the soft vascular tissue which lies external to the dura mater. In broth inoculations from the heart blood he obtained a pure culture of a micrococcus indistinguishable from that found in the external part of the dura. The subdural injection of a cultivation of this organism into a rabbit was followed by a rapidly-spreading

<sup>1</sup> *Brain*, 1903, p. 94.

palsy. The animal was killed, and on histological examination changes in the nervous system of the kind produced by toxins were discovered. The micrococcus was demonstrated in the dura mater of the rabbit and isolated in pure culture from its blood, but was not found in the nervous structures or in the pia arachnoid.

In our case, as already mentioned, a broth inoculation from the heart blood was sterile, and the examination for organisms of the epidural tissue by methylene blue proved negative.

#### EXPLANATION OF PLATE 17.

FIG. 1.—From the right facial nucleus (Nissl).

FIG. 2.—Right facial nerve (Busch).

FIG. 3.—Two anterior horn cells from the first lumbar segment (Nissl). That on the right is the only cell in this section which presented a fairly normal appearance. The cell on the left shows well-marked chromatolysis. Many cells at this level showed changes more striking than depicted in this photograph, in which, however, the contrast between a normal cell and one in which there is very evident breaking up of the Nissl bodies is well seen.

FIG. 4.—Cells from Clarke's column at the level of the first lumbar segment (Nissl).

FIG. 5.—External popliteal nerve (Busch).

FIG. 6.—Tibialis anticus muscle (Busch), showing one fibre in which there is a well-marked fatty degeneration, several others in which these appearances are less obvious.

(The writer's thanks are due to Mr Richard Muir, of the University Pathological Department, for the photographs represented in this plate.)

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## Abstracts

### ANATOMY.

**ON THE AREAS OF THE AXIS-CYLINDER AND MEDULLARY (149) SHEATH AS SEEN IN CROSS SECTIONS OF THE SPINAL NERVES OF VERTEBRATES.** H. H. DONALDSON and G. V. HOKE, *Journ. of Comp. Neurol. and Psychol.*, Jan. 1905, p. 1.

In this paper the authors have endeavoured, and with commendable success, to show that in an accurate transverse section of a spinal nerve, the area occupied by the medullary sheath is, on

an average, practically equal to that occupied by the axis-cylinder. But, what is perhaps more interesting still, they likewise point out that this interesting relationship exists in all the five great vertebrate classes,<sup>1</sup> and also that it holds good for (1) nerves having different diameters; (2) those afferent or efferent in function; (3) those taken from various parts of the body; (4) males and females; (5) different seasons; (6) different ages; and (7) animals active and those slow in their movements.

On account of the great delicacy of the operations involved in making the requisite measurements, it was necessary to find a fixative which had the least effect in altering the dimensions of the nerve fibre, and for this purpose the authors found osmic acid to be the most suitable agent. It was also essential to make a judicious selection of suitable fibres, only those with a circular outline being employed, while immature fibres and the very small ones found in the *rami communicantes* were not examined.

The method of measurement adopted may be briefly described as follows. The diameters of the whole fibre and of the axis-cylinder were first of all measured by means of an ocular micrometer, and from these the corresponding radii in  $\mu$  were readily obtained. The formula employed to calculate the areas of the fibre and of the axon was  $\pi r^2$ , the value of  $\pi$  being taken as 3.14. Owing to the great variability in the diameters of the different fibres, it was necessary to take the square of the average radii in twenty or more nerve fibres, and multiply these results by 3.14 in order to obtain the average areas in sq.  $\mu$ . On subtracting the area of the axon from that of the fibre, the area of the medullary sheath was at once ascertained.

It was, in the next place, necessary to indicate a standard to go by, and for this purpose the authors adopted an "ideal area," which was obtained by dividing the total area of a transverse section of the fibre by 2—thus allocating one-half to the sheath and the other half to the axis-cylinder. From an examination of 1540 fibres it was found that the average percentage deviation of the medullary sheath from the above standard only amounted to +0.45 per cent., that is to say, the area occupied by the sheath was as a rule slightly more extensive than that of the axon. It ought to be noted, however, that extreme deviations from this standard, both in the *plus* and *minus* direction, were found throughout all the vertebrate classes, and these had to be taken into account in calculating the grand average, a fact which enhances the value of the results obtained in this interesting investigation.

JOHN CAMERON.

<sup>1</sup> The Acrania and Cyclostomata are excluded by the fact that in these the nerve fibres do not possess a medullary sheath.

**THE TECHNIC OF NISSL'S METHOD.** (*Sulla tecnica del metodo* (150) *di Nissl*.) E. LUGARO, *Monitore Zoologico Italiano*, No. 1, 1904.

NISSL's methods for the coloration of the chromophile substance of the nerve cells have been modified in innumerable ways by various workers. Indeed, the original methylene blue method, as Lugaro justly remarks, is now used by no one excepting Nissl himself and his pupils. In this paper the author, after alluding to the grounds for some of the main departures from the original technic, expresses the opinion that the ideal method would be one in which there was a progressive staining of a strictly selective character, not followed by any process of decolorisation, but by fixation of the stain before dehydration, clearing, and mounting. He describes a staining process of this nature which he has devised. It yields preparations in which the chromophile substance of the nerve cells stands out against an absolutely colourless ground. The various steps are as follows:—

(1) Fixation for 48 hours in a solution of 5 per cent. pure nitric acid in absolute alcohol.

(2) Absolute alcohol, xylol, paraffin.

(3) Fixation of the sections (6 to 8  $\mu$ ) upon slides with the aid of distilled water.

(4) Removal of the paraffin by passing through xylol, alcohol, and then distilled water.

(5) Staining for several hours in a weak solution of toluidin blue in water (1 in 2000, or 1 in 3000).

(6) Washing in distilled water for a few seconds.

(7) Fixation of the colour in a 4 per cent. solution of ammonium molybdate for two or three minutes.

(8) Washing in distilled water, dehydration, clearing in xylol, mounting in balsam.

W. FORD ROBERTSON.

## PHYSIOLOGY.

**NOTES ON THE DEVELOPMENT OF THE SYMPATHETIC** (151) **NERVOUS SYSTEM IN THE COMMON TOAD.** WALTER C. JONES, *Journ. of Comp. Neurol. and Psychol.*, March 1905, p. 113.

THE author describes the sympathetic as arising from cells in the mesoblast, but of unascertained source. From their proximity to the nerves he considers they may be really of epiblastic origin.

Between the vagus ganglion and the second spinal nerve the sympathetic arises directly by the aggregation of these scattered cells into a cellular cord. They are in contact from the earliest stages with the fibres of the first and second spinal nerves, thus

differing somewhat from the process in mammals described by Paterson.

Posterior to the second spinal nerve the origin is more complex. A ridge of cells appears alongside the aorta; the top of the ridge is differentiated to form the sympathetic cord while the remainder disappears, leaving the cord free, save its connections with the collateral sympathetic and with the spinal nerves. The ganglia and commissures arise directly from the cord by enlargement or reduction of the ridge. This contrasts with Balfour's observation on elasmobranchs, where he finds the ganglia arising independently of each other and the commissures appearing as later outgrowths from the ganglia.

As the cord is removed from its primary contact with the nerves, fibrous connections remain which constitute the rami communicantes.

A short digest of the literature is given.

J. H. HARVEY PIRIE.

## PSYCHOLOGY.

**A STUDY OF THE EMOTIONS.** W. H. B. STODDART, *Brain*, (152) Winter 1904, p. 509.

### I. *The physical basis of the emotions.*

This is an endeavour to establish the two propositions:—

(1) That an emotion is a sensation-complex, its component sensations being entirely derived from a complex, usually involuntary, motor response to a percept or idea of some situation or incident.

(2) That the motor paths subserving the function of this involuntary response are those of the primitive nervous system, viz.: the cortico-rubral system of fibres and the rubro-spinal tract.

The view expressed in proposition 1 is that set forth by Professor James in his "Principles of Psychology." An emotion consists of a number of sensations derived from the activities of certain muscles (voluntary and involuntary), and glands (sudorific, lachrymal, etc.). The activities of involuntary muscles give rise to local flushings and pallors. These various activities which give rise to the emotions are also responsible for the expression which indicates to others the nature of the particular emotion experienced.

The argument is developed that it is not the emotion which gives rise to its expression, but the expression which gives rise to the emotion. The movements which constitute the expression

are remnants of instinctive acts which, in the past history of the race, have served a useful purpose, when the organism was placed in such a situation as now merely arouses an emotion.

The premises on which the second proposition rests are:—

(a) That lesions of the Rolandic area cause on the opposite side paralysis of voluntary movement, but not of emotional expression.

(b) That lesions of the thalamic region cause on the opposite side paralysis of emotional expression, but not of voluntary movement.

It follows that the motor element of emotion is subserved by some motor tract which crosses from the mesencephalon to the opposite side of the cord, most probably the rubro-spinal bundle of Monakow. The cortical portion of this motor system consists of the cortico-rubral fibres described by M. and Mme. Dejerine. This cortico-rubro-spinal motor system is at least the main representative of the primitive motor system, by which in the lower vertebrates all motor impulses are transmitted.

The neural process, which takes place when an emotion occurs, is then as follows:—

Starting from the stage at which a sensation is registered in one of the projection areas, or a percept or idea formed in one of the association areas of the cortex, an impulse is transmitted to the red nucleus by way of the cortico-rubral fibres, thence to the large motor cells of the lowest level by way of Monakow's rubro-spinal (and presumably rubro-bulbar) fibres of the pristine motor system, and thence to the muscles of expression. Contraction of these muscles upon their spindles effects the transmission of muscle-sensations to the cortex by way of the ordinary sensory paths; and it is the particular combination of these sensations among themselves and with vaso-motor sensations which determines the given affective or emotional tone.

## II. *The pathology of the emotions.*

This section deals with the conditions in which there is excess or deficiency of emotional reaction.

Excess of emotional reaction occurs in all conditions in which the pyramidal tract is more degenerate than the pristine motor system, because motor impulses then tend to be transmitted by the less degenerate cortico-rubro-spinal system. This state of affairs obtains in hemiplegia, general paralysis, alcoholic insanity, epileptic insanity, and probably in imbecility; all such patients are apt to react emotionally to unimpressive stimuli.

Maniacal patients are also excessively emotional for another reason, viz. that the motor neurons are in a constant state of irritability, and that minimal stimuli therefore suffice to induce discharge of the cortico-rubral neurons.



The emotional outbursts of paranoiacs and patients suffering from hallucination are due to an excess of perception; and morbid fears to an abnormal tendency of the pristine motor system to react to some particular percept.

Deficiency of emotional reaction is due, in confused and stuporose states, to the associated anæsthesia; the component sensations of a percept being unsatisfactorily served up to the sensorium. In secondary dementia, senility, myxœdema, and cretinism, the emotional defect is due to partial or complete imperception.

The deficiency of emotional reaction in melancholia is the result of fixation of the muscular system. The emotional defect of katatoniac stupor is similarly accounted for.

The conclusions arrived at in this section are summarised as follows:—Excess or defect of emotional reaction may be dependent upon excess or defect of sensation, or upon excess or defect of perception. Excess of emotional reaction may also depend upon an abnormal tendency of motor impulses to be transmitted *via* the pristine emotion-arousing motor tracts of the nervous system; and defect of emotional reaction may further be due to fixation of emotion-arousing musculature.

AUTHOR'S ABSTRACT.

**ON FLIGHT OF IDEAS: A CRITICAL EXAMINATION OF H. (153) LIEPMANN'S WORK "ON FLIGHT OF IDEAS, DEFINITION AND PSYCHOLOGICAL ANALYSIS. (Ueber Ideenflucht, etc.)** By E. STORCH (of Breslau), *Monatsschr. f. Psych. u. Neur.*, Jan. 1905, p. 38.

LIEPMANN tries, in the work referred to, to solve the question of the difference between logical thought and flight of ideas. Storch agrees with him in his view that the answer cannot be found in the nature of the principles of association involved; the laws of association are an inadequate explanation of logical thought. In flight of ideas, while the attention may have a certain amount of energy, it has little constancy; according to Liepmann the essence of logical thought is to be found in the constant direction of attention under the guidance of a leading idea or *Obervorstellung*. Attention grasps in one act a definite complex of ideas, and in the ensuing steps of logical thought the attention is directed to the elements of that complex, which thus forms the bond between them. Storch points out the inadequacy of this explanation, for example in the case of scientific thought, and insists that there is no objective criterion for logical thought. The subjective characteristic of logical thought is that each step

appears as the willed sequel of the preceding state of consciousness; we cannot replace the goal-idea to which we are striving by the *Obervorstellung* of Liepmann. Storch then proceeds to elaborate a psychophysical hypothesis to elucidate the difference between logical thought and flight of ideas.

C. MACFIE CAMPBELL.

**THE PSYCHOLOGY OF FORGETFULNESS IN INSANITY AND**  
(154) **NERVOUS DISEASE.** Prof. A. PICK, *Archiv. f. krim. Anthropol.*  
*u. Kriminalistik.*, Bd. xiii., p. 251.

IN a short paper under the above title, Prof. Pick reviews the work which has been done in the field of minor mental lapses, ellipses, the formation of false memories (the *transitivism* of Wernicke, etc.), by Wernicke, Janet, and others, and particularly that of Freud, published in the *Monatsschr. f. Psych. und Neurol.*, iv. Heft. 6 and 7, on "The Psychical Mechanism of Forgetfulness," and as a separate pamphlet, entitled "The Psychology of Everyday Life," published in 1904, and abstracted in this Review in the June number of last year. Prof. Pick supports the views of Freud—which have not, he says, been sufficiently appreciated—in which the importance of affective disturbance in the causation of their phenomena is insisted upon. Freud's observations, however, have been mainly concerned with the hysterical, and in this paper Pick examines their occurrence in the insane. Notes of one or two interesting cases are given and carefully analysed. The subject is one of universal interest and much importance, as investigations of this kind cannot but throw light on many obscure problems in normal psychology.

R. CUNYNGHAM BROWN.

## PATHOLOGY.

**INVESTIGATIONS ON THE YELLOW PIGMENT OF NERVE**  
(155) **CELLS.** MARINESCO, *Revue de Psychiatrie et de Psychologie*  
*Experimentale*, fév. 1905.

**ON THE PRESENCE OF A SPECIAL NETWORK IN THE**  
(156) **PIGMENTED REGION OF NERVE CELLS.** (Sur le présence  
d'un réseau spécial dans la région pigmentée des cellules  
nerveuses.) MARINESCO, *Journ. de Neurol.*, mars 5, 1905, p. 81.

THE author in this paper makes another interesting contribution to our knowledge of the origin, chemical composition, and function of the pigments which are found in nerve cells.

Dealing principally with the yellow pigment, he first mentions

the explanations which have been offered in regard to its production and chemical composition, and then he goes on to give us the results which he has obtained recently by means of the methods of Marchi, Nissl, and Cajal, and by the employment of Sudan.

These methods show that the yellow pigment found in the nerve cells consists of granulations lying in an amorphous yellow ground-substance.

The granulations are of various sizes, and, as a rule, the smaller granulations are found in the larger cells; small and large granules are rarely found in the same cell.

They also vary in colour, and even with the same method—Nissl or Sudan—tints from light yellow through yellow ochre to dark brown or black may be seen. The darker tints become more common as age advances.

The yellow pigment is generally situated at the periphery of the cell, but it is sometimes found around the nucleus.

In the cells of the spinal ganglia another substance—melanine—is often present. This substance exists in the form of larger granules which are stained dark brown or black by Sudan; it is more commonly seen in the cells of these ganglia than is the yellow pigment.

Both these pigments are present in greater quantities in the lower ganglia; they are also found in the cells of the sympathetic system, but not in those of the jugular or ophthalmic ganglia. They are absent from the cells of young people.

Frequently a large amount of pigment is found in the cone of origin of the axis-cylinder, and Marinesco suggests that this may be explained by the active chemical processes, which go on where the axis-cylinder fibrils spring from the intracellular fibrillary network, and which lead to morphological modifications of the chromophile elements, and to the formation of pigment.

The use of Sudan has enabled the author to show that the pigment is sometimes eliminated from the cell, and it may then be seen lying in the small vessels and in their adventitia, and also in the neuroglia cells. This is a strong argument against the view that the pigment furnishes a nutritive material to the nerve cell.

The factors which determine the appearance of the pigment are—(1) advanced age; (2) disturbances of the nutrition of the cell; (3) pathological degenerative conditions.

The pigment probably constitutes a normal product in the life of the cell, and it increases in quantity and alters in character as age advances. It may appear in the fundamental substance of the nerve cell as the product of cellular activity, possibly regressive, or it may result from the disintegration of the chromophile substance.

With regard to its chemical composition, the author expresses the opinion that, although the yellow pigment contains some fat, it does not consist entirely of fatty matter, and it must not be considered to be produced by a fatty degeneration of the chromophile elements. He does not admit that it is a lipochrome, as Rothmann and others have suggested.

In his second paper Marinesco deals with the appearances which are obtained by using Cajal's silver nitrate method for staining the fibrils in the nerve cells. He has found that the fibrils undergo some important modifications in the portions of the cells in which the pigment is deposited.

Using pieces of tissue which have been fixed in alcohol according to Cajal's method, it is easy to see a dark streak in the nerve cells, which, by a higher magnification, is found to consist of a coarse fibrillary network, with meshes of various sizes, and stained nearly black. These meshes contain a brown fundamental substance in which lie the pigment granules.

Generally the network and the pigment are situated at the periphery of the cell, but they may lie around the nucleus.

It has been found that the advanced age and the various pathological processes which favour the formation of pigment also lead to the appearance of this network.

With regard to its origin, Marinesco has come to the conclusion that the darkly stained network is the result of a chemical transformation of the ordinary fibrillary network. This hypothesis suggests that with this change in the fibrils there may be some alteration in their conducting power for nervous stimuli, and that the specific intensity of the nervous waves may be diminished in these thickened fibrils of the nerve cells, as in the case of electric currents along conductors of different calibre.

It is possible that the nerve energy in old people may be considerably reduced by such a condition. In pathological conditions also, in which these fibrils are thickened, it may be that this thickening is followed by an interference with the transmission of the nervous waves.

R. G. Rows.

**THE PLASMA CELLS OF GENERAL PARALYSIS.** (*Les cellules (157) plasmatique de la paralysie générale.*) DE BUCK, *Journ. de Neurologie*, March 20, 1905, p. 101.

In an earlier paper the author adopted the view that the plasma and other cells forming the periarthritis of general paralysis were derived from the blood. This is now discarded in favour of the theory that the plasma cells are fibroblastic in origin, and are derived from both endothelium and adventitia. They also have

a certain amount of migratory power, and can invade the adjoining nervous tissue.

The clasmatocytes of Ranvier, and the "Mastzellen," are only more differentiated forms than the plasma cells. All are of fibroblastic origin. Frequently they (the plasma cells) are full of metachromatic granules or vacuoles, and the nucleus is strongly chromatic. Probably they have no phagocytic properties; more likely they help in the formation of "anticorps." In general paralysis they may help the neuroglia in the sclerotic process. The author joins in the opinion that this disease is due primarily to a vascular lesion.

DAVID ORR.

**CALCIFICATION OF THE FINER CEREBRAL VESSELS, WITH (158) REMARKS UPON ITS CLINICAL SIGNIFICANCE.** Professor PICK, Prague. (A Translation.) *American Journal of Insanity*, No. 3, 1905, p. 417.

THIS paper is an addition to the work already done by the author on the subject, and the only new points dealt with are the calcification of the elastic fibres and the calcospherites. Paraffin series of non-decalcified vessels are prepared, drawn under a low power and afterwards decalcified and stained.

The process begins in the muscularis, the lime accumulations forming a homogeneous thick ring and progresses centripetally. In the first stages the deposit takes place between the muscle cells. The elastic fibres of the media are the first to become calcified, and probably as a result of pressure the muscle cells are destroyed and themselves undergo calcareous impregnation. Proliferation of the intima takes place, and at times precedes the calcification process. Enclosed in the proliferation sometimes there are seen granules stained with hæmatoxylin. These are evidently colloid balls which are the forerunners of a calcification.

In decalcified and stained specimens there are large dark nuclei surrounded by a homogeneous space. These are sometimes closely united and form large masses. The author regards them as some sort of inorganic formation, and is inclined to classify them with the so-called calcospheres. They are not very numerous and are probably formed in places in which the lime salts are suspended in stagnating blood plasma. Actively flowing blood hinders their formation, hence they are only found where total obliteration of the vessel has taken place, or at points near the obliterated part.

In the clinical part of the paper the author draws attention to the association of the above calcification process with tetany.

DAVID ORR.

**SYSTEM LESIONS IN THE POSTERIOR COLUMNS IN GENERAL**  
**(159) PARALYTICS, AND THEIR BEARING ON THE POINT OF**  
**ORIGIN OF TABES DORSALIS.** ORR and Rows, *Brain*,  
Winter 1904, p. 460.

EXAMINATION of the posterior columns of the cords of several general paralytics has shown that the degeneration of the fibres has always begun at a definite spot, and has spread from that spot to the terminations of the fibres. The cases examined exhibited the lesion in an early phase of degeneration before any sclerosis had appeared, and on this account all the tissues were examined by the Marchi method.

The important points which were noticed in the case which has been described in detail were:—(1) Degeneration of the internal division of the sensory roots in their intramedullary path, commencing at the point of entrance into the cord, and following the usual anatomical course of the fibres in the cord. (2) The collaterals and terminals of the sensory system, which pass into the grey matter, share in the degenerative process. (3) Complete integrity of the outer division of the sensory root, Lissauer's tract. (4) The long fibres which were affected in the root-entry zone in the lumbar region could be followed into Goll's column in the cervical region.

An examination of the posterior roots connected with the various segments often failed to show any lesion, and when a lesion was present it was so slight that it could not possibly have been the cause of the degeneration within the cord.

The absence of any degeneration in the roots may be explained by the facts regarding the atrophy of the fibres after section of the posterior root, which have been determined by Köster. He has shown that the cells of the posterior ganglia do react after section of the posterior root, but at a much later period than after section of the peripheral process. The fibres belonging to the cells which pass on from cell-reaction to cell-degeneration undergo a Wallerian degeneration. The others, and these form the great majority, undergo a slow atrophy. Such a simple atrophy has been recorded by various observers in the posterior roots of tabetics.

The point at which the fibres were attacked was found to correspond with the point at which they entered the cord, i.e. at Obersteiner's ring. At this point an important change takes place in the histological structure of the nerve fibres. The fibres here lose their neurilemma sheath, and their myeline sheath is either entirely absent or is extremely thin.

The bare axis-cylinder is therefore exposed to any noxious agent which may be carried to it by the lymph stream.

In connection with this, it is important to remember the work of Homén, D'Abundo, Marie and Guillain, and others. These observers have found that the lymph flows towards the cord in the perineural sheath, and, in the cord, it passes forwards and upwards towards the central canal.

The degeneration of the sensory fibres in these cases, then, has commenced where the fibres have lost their neurilemma sheath and where the unprotected axis-cylinder has been attacked by some toxin which has reached them by the lymph stream. This toxin has produced a primary degeneration of the nerve fibres, which is characterised by a progressive atrophy of the myeline sheath, while the integrity of the axis-cylinder is maintained for some time. Then, either because of the virulence of the poison or its long continued action, a secondary degeneration supervenes, and the fibre degenerates from the point of injury to its termination, *i.e.* by a centrifugally spreading degeneration.

This is entirely opposed to the view of Nageotte, who maintains that the fibres are injured by a transverse neuritis of the posterior root, and that then a Wallerian degeneration begins at the distal extremity and spreads backwards towards its trophic centre.

But not all the fibres of a given root and not all the roots of a given cord have been affected to an equal extent. This condition has sometimes rendered it a little difficult to recognise the systemic character of the lesion.

Such selection has, in all probability, been determined by some developmental anomaly or by other predisposing causes—the most important among which would be syphilis—rather than by any special selectivity on the part of the poison.

AUTHORS' ABSTRACT.

#### ON THE SPINAL CORD DEGENERATIONS IN ANÆMIA.

(160) J. MICHELL CLARKE, *Brain*, Winter 1904, p. 441.

THE paper deals with cases in which spinal cord changes follow on severe anæmia, that is to say, are distinctly secondary to it. Five cases are reported at varying stages. In two the lesions of the cord gave rise to no symptoms during life. In the first case the patient died of pneumonia shortly after admission to hospital, and in this the earliest changes were found in the cervical region of the cord.

Case 2 was one of profound anæmia of 3½ years' duration; paræsthesiæ and paresis of legs after 2 years; recovery from anæmia; death from gangrene of lung following pneumonia; post-mortem stationary changes in cord. The interest of this case, which was under observation throughout, lies in the fact of recovery from the anæmia, and in the apparently stationary char-

acter of the cord-lesions. In the other three cases there was more extensive degeneration of the cord than in the foregoing.

The series of cases shows that the spinal degenerations secondary to anæmia begin in the posterior columns, and in the cervical and upper dorsal regions of the cord. In cases which die from any cause in an early stage, the lesion of the cord is limited to these parts. In more advanced cases the posterior columns are affected almost throughout the cord, but most extensively in its upper part (cervical region). Though the changes are fairly regular, they do not exactly map out special tracts. The posterior roots are unaffected. Though the appearances suggest, in some instances, that the degenerations bear some relation to the vascular supply, this is not a constant feature. In the most marked cases, patches of degeneration occur also in the lateral columns: in the dorsal or upper dorsal regions first. Marchi-stained sections show a wider distribution of lesion than is indicated by the Pal-Weigert stain. The finer changes in the degenerated areas are practically identical with those described by Dr Risien Russell in cases of "subacute combined degeneration" of the cord. The primary change seems to be a parenchymatous degeneration of the nerve fibres; increase of neuroglia and vascular changes are secondary to this. There may be no clinical symptoms, and moreover, the pathological changes may not be found until after the cord has been hardened and stained. Clinically the symptoms are especially various paræsthesiæ, first appearing in the feet and legs; objective loss of sensation may occur in the later stages, but is not marked; paresis, first in the legs; muscular twitchings; reflexes are retained; no ataxy; no incontinence of urine.

These changes in the cord occur in forms of anæmia due to blood destruction, not only in that form known as "pernicious anæmia"; it is suggested that they may be due to a toxin, set free in the course of a pathological hæmolysis.

The following points would seem to separate this group from other similar degenerations:—

1. Severe anæmia distinctly preceding any evidence of lesion of the cord.
2. The relative slightness of clinical symptoms.
3. Restriction of lesion to posterior columns of cord in most cases.
4. Difference in distribution of lesions, especially from "diffuse" or "subacute combined degeneration" of cord; from mild cases of the latter by the less definite limitation of the lesion to the neuron systems of the cord, and from more severe cases by the far less extent of degeneration.

Difficulty of differentiation occurs chiefly in cases which have been described, in which extensive cord changes coincide



with severe anæmia; it is suggested that provisionally such cases might be left for further investigation, and a first classification of the forms of degeneration of the cord associated with anæmia made by separating into two distinct groups, (1) the class of cases described in this paper, and (2) those of "diffuse" or "subacute combined degeneration" of the cord, which follow the course given by Drs Russell, Batten, and Collier. AUTHOR'S ABSTRACT.

### CLINICAL NEUROLOGY.

**CRITICAL STUDY OF JUVENILE TABES.** (*Étude critique sur le (161) tabes infantile juvénile.*) E. HIRTZ and H. LEMAIRE, *Rev. Neurol.*, March 15, 1905, p. 265.

THE authors have collected, from the records of medical literature, 46 cases of juvenile tabes. The chief symptoms of these cases are given in tabular form, and a critical review of the disease is added.

The authors also record one case of juvenile tabes. The chief symptoms were: gastric crises, Argyll-Robertson pupil, inequality of the pupils, diminution of vision (but optic discs normal), loss of the tendo Achillis reflexes, knee-jerks present, lymphocytosis of the cerebro-spinal fluid.

The authors point out that no post-mortem examination of a case of juvenile tabes has yet been recorded.

The disease may commence with urinary troubles, or with shooting pains, or with failure of vision. In two cases gastric crises have been the first symptoms. The most frequent functional affection, in the preataxic period of juvenile tabes, is incontinence of urine.

In the 46 cases, tabulated by Hirtz and Lemaire, bladder symptoms (paresis, incontinence or retention) were present in 23, and optic atrophy was present in 10 cases. As regards syphilis, in these 46 cases there was evidence of hereditary syphilis in 26, hereditary syphilis was probable in 8, doubtful in 2. There had been syphilitic infection (acquired syphilis) at an early age in 4 cases. There was no evidence of syphilitic disease in 5. Thus there was evidence of syphilis (hereditary or acquired) in 30; also in 8 cases hereditary syphilis was probable.

R. T. WILLIAMSON.

**TWO CASES OF SYPHILITIC DISEASE OF THE CERVICAL (162) SPINE.** FRANK R. FRY, *Journ. Nerv. and Ment. Dis.*, Feb. 1905, p. 101.

FRY records two cases characterised by marked rigidity and pains in the neck. The head was held rigidly in the middle line, with

chin depressed. The pains were very severe, coming in paroxysms, worst at night, reaching their maximum at certain points. In the first case, they had the distribution of a cervico-brachial neuralgia, chiefly of the right side; while, in the second case, the distribution was that of cervico-occipital neuralgia of the left side. No objective sensory loss was found in either case, but there was numbness about the shoulder in the first case, and about the ear in the second. Both patients were much emaciated and reduced in strength by the pain, sleeplessness, etc. Both made a practically complete recovery under anti-syphilitic treatment (mercurial inunction and iodide of soda by mouth). In both, after satisfactory manipulation of the neck became possible as a result of this treatment, a deep swelling, very sensitive to pressure, was found in the neck behind the transverse processes of the fourth and fifth cervical vertebræ; the swelling and tenderness alike disappeared almost entirely under treatment.

The first case had probably had syphilis six years before; in the second case, there was no history or evidence of syphilis, and the history of onset after he had "caught cold" would point rather to a rheumatic origin. The author, however, believes that both cases "comport with what might be called a type of cases, a sub-class of cervical spinal syphilis, characterised by a rigidly stiff neck, with one or more points of great tenderness on deep pressure, severe neuralgic pains, often not very sharply localisable, no objective sensory changes, no paralysis, recovery on anti-syphilitic treatment." Records show that syphilis of the spine is a rare affection.

A. W. MACKINTOSH.

**THE CYTO-DIAGNOSIS OF GENERAL PARALYSIS.** (Zur Frage (163) der Zytodiagnose der progressiven Paralyse.) FISCHER, *Prager med. Wochenschrift*, xxix., No. 40, 1904, S. 513.

FOUR cases of general paralysis gave varying results on the cytological examination of the cerebro-spinal fluid: one, characterised by an exceedingly sparse lymphocytosis, was associated post-mortem with thickened meninges; another showed a converse condition.

S. A. K. WILSON.

**CERVICAL PACHYMEINGITIS.** (Zur Frage der Pachymeningitis (164) interna chronica cervicalis hyperplastica.) FISCHER, *Zeitschr. f. Heilk.*, Bd. xxv., H. 10, 1904.

ACCORDING to the classical description by Charcot, the chief feature of this disease is a chronic hypertrophy of the meninges

of the cervical cord, associated with grave alterations in the cord itself, the latter being secondary to the compressing effects of the former. Adamkiewicz believed that both were the result of chronic infection. Others hold that the cord degeneration is attributable to vascular degeneration.

The opinion expressed in this paper, based on the pathological findings in two cases (not diagnosed during life), is to the effect that malnutrition, subsequent to disease of the blood-vessels, is the probable cause, both of the medullary degeneration and the meningeal changes. In each case the cervical cord was the seat of a chronic hyperplasia of the dura mater, and to a less extent of the other meninges, which was not found below the upper dorsal segments, but extended headwards into the medulla. Between the dura and the arachnoid was a conglomerate of endothelial cells, some showing hyaline degeneration, others calcification; in addition were to be seen many concentric connective tissue bodies, evidently derivatives of vascular degeneration. The greater part of the dural thickening was due to changes round diseased blood-vessels.

In the cord, however, little, if any, chronic pathological alteration was noted, and the explanation offered is that the cases were incipient ones. What areas of softening there were, were acute, and showed little resulting secondary degeneration. A correlation of the clinical symptoms and the pathological findings is not given.

S. A. K. WILSON.

**TRANSVERSE SPINAL SCLEROSIS.** (*Sclérose médullaire transverse, (165) segmentaire, dorsolombaire gauche, métatraumatique. Forme clinique curable.*) RÉVILLIOD, *Nouv. Icon. de la Salpêtr.*, jan., fév, 1905, p. 17.

THE subject of the interesting case here reported was a healthy woman who slipped one day in going downstairs and sustained a fracture of the two malleoli on the right side, with a subluxation of the right astragalus. Fourteen months later she came under observation with a left hemiparaplegia and a hemidysæsthesia, much more marked on the right side than on the left; in other words, with a complete and almost flaccid paraplegia limited to the left lower extremity, there was loss of cutaneous sensation on the right lower extremity, and at the same time a crossed thermo-anæsthesia, the left leg recognising cold, but not heat, the right heat, but not cold.

Anatomically one must postulate a lesion involving the left half of one of the dorso-lumbar segments, and including:—

1. The left crossed pyramidal tract,

2. The centripetal path for heat sensation from the left lower extremity (central grey matter).
3. The cutaneous and cold sensation afferent tracts from the right, which therefore probably cross in order to reach the site of the lesion on the left side.

The cord below this segment was probably normal, as the patellar reflexes were exaggerated and sphincter function unimpaired.

A year later the clinical picture was to a certain extent modified, in that the anæsthetic right leg became less so, the sensation of cold returning, whereas the left leg began to recover some power, at the same time becoming more anæsthetic, and beginning to lose also the sensation of cold. Apparently the original unilateral transverse myelitis (for this is the author's diagnosis) began to diffuse itself more posteriorly, crossing the middle line.

Six months later a sudden complete motor and sensory paralysis of the legs, associated with severe pains, indicated a sudden development of a hæmatorachis. Its absorption in the course of a few days was followed by a rapid and steady recovery of all motor and sensory function in the affected limbs, till at present there remains practically no trace of the disease. In the writer's opinion the case is a valuable illustration of the action of peripheral trauma on medullary centres, and his contention is supported by various citations from the literature. The details of the treatment, carried out over many months, and varied from time to time, are recorded with praiseworthy fulness, and are justified by the event.

S. A. K. WILSON.

#### **THE FALSE LOCALISING SIGNS OF INTRACRANIAL TUMOUR.**

(166) JAMES COLLIER, *Brain*, Winter 1904, p. 490.

It is only possible in this abstract to refer briefly to some of the conclusions arrived at in this important paper. False localising symptoms constitute a common cause of error in localising tumour of the brain. This paper is based upon 161 consecutive cases examined in the Laboratory of the National Hospital, and most of which had during life come under the author's personal observation.

The author describes two cases to illustrate what he means by *the false portent of late localising symptoms*. The first case was that of a man who, fifteen months after the first appearance of his symptoms, developed local signs indicative of a lesion in the left posterior fossa. A glioma of the left pre-frontal region was found post-mortem. These pseudo-localising symptoms were explained by the pushing downwards and backwards of the left lateral lobe of the cerebellum into the foramen magnum.

The second patient, in whom there were signs of a left-sided cerebellar lesion, after twelve months developed Jacksonian attacks confined to the left arm. The convulsions afterwards became general, with loss of consciousness. At the post-mortem, six months later, a tumour of the cerebellum was found, with great ventricular distension, the dilatation being most marked in the central region of the right ventricle.

False localising symptoms were present in 12·5 of the 161 cases, being more common in supra- than in sub-tentorial growths.

Anosmia and nerve deafness were never met with apart from direct implication of the olfactory tract and auditory nerve, and the author is of opinion from extensive observations that there is not the least evidence to show that an olfactory or auditory neuritis comparable to optic neuritis ever occurs.

In two cases, hemianopia occurred as a false localising symptom. In both instances there was paralysis of the fifth nerve on the same side, the symptoms probably depending upon indirect pressure on the middle fossa by a tumour in the cerebrum of the same side.

Paralysis of the sixth nerve was met with in twelve cases of supra-tentorial tumour. The author is unable to accept the view commonly advanced to account for the frequency of paralysis of this cranial nerve, viz., that its long course especially predisposes it to pressure; he advances the view that the probable explanation is a pressing downwards of the brain stem and cerebellum by the supra-tentorial growth, direct traction on the nerve taking place. The other nerves do not suffer so frequently, because they are directed transversely.

Third nerve paralysis was present in two cases.

The fifth nerve was twice affected, and in both instances there was associated hemianopia.

In two cases there was facial palsy. The author has no explanation to offer for slight weakness of the lower face unaccompanied by diminished electrical excitability, which is of common occurrence in supra-tentorial growths.

In no cases were the ninth, tenth, eleventh or twelfth nerves affected. Occasionally a cranial nerve palsy occurring as a false localising sign has been seen to disappear.

In two cases of cerebellar tumour of long standing and in a case of glioma of the pons in which considerable ventricular distension was found post-mortem, local convulsions were observed. When localised or generalised convulsions only appear long after the general signs of intracranial growth, they are to be disregarded as a localising sign.

Bilateral spastic paresis may occur in connection with old-standing ventricular distension, and is probably dependent upon

cortical wasting due to the dilatation of the ventricles. The distinction between this condition and spastic paresis dependent on a lesion of the brain stem may be difficult.

Cerebellar symptoms dependent upon a pushing downwards and backwards of the cerebellum into the foramen magnum are, in the author's experience, not unfrequent.

Sub-tentorial growths, with the exception of some cases of pontine glioma, almost always produce early localising symptoms. Conversely, a tumour which causes marked general symptoms, but no local signs for many weeks, may be located above the tentorium.

Meningitis was only present in one case of this series.

In two cases a vascular lesion occurred at some distance from the growth.

Local spreading oedema in the neighbourhood of, it may be, a small tumour, may give rise to severe, it may be rapidly fatal, symptoms.

Deposits secondary to a primary cerebral growth occurred in two cases.

Three patients presented shooting pains in the limbs, loss of the deep reflexes in the lower extremities, and ataxy of the legs—symptoms probably referable to a posterior column degeneration.

Localising symptoms may be concealed, as in two cases of occipital tumour, in which the patient had become blind from optic neuritis, the hemianopia which must have resulted from the situation of the tumour being in consequence lost as a localising sign.

Cerebellar disease may be difficult to diagnose in cases of long standing, in which the patient has lost power of voluntary movement and of attending intelligently to command.

EDWIN BRAMWELL.

**ON RECURRENT ATTACKS OF EUPHORIA IN A CASE OF (167) CEREBRAL TUMOUR.** (Ueber anfallsweise auftretende euphorische Stimmung bei Hirntumor.) PICK, *Wien. klin. Wchschr.*, Jan. 5, 1905, p. 25.

A TYPICAL case of Jacksonian epilepsy from cerebral tumour had from time to time, and independently of the cortical fits, "attacks" of euphoria, which the patient himself described spontaneously as a "sensation of bliss"; during these periods he seemed to have incomplete control over his thoughts, but apart from the facial expression corresponding to the mental exaltation there was nothing to be observed. In the intervals his mood was depressed, and quite the converse of the euphoria.

References in the literature to analogous phenomena are exceedingly scanty, and the writer is inclined to reserve his opinion on their significance pending further investigation. It may be remarked that an identical sense of well-being, so frequent in phthisics and general paralytics, must theoretically be traceable to some altered cerebral function, on the localisation of which the case here reported (clinically a tumour in the neighbourhood of the ascending frontal convolution, probably still more anterior) ought to shed some light. Unfortunately (though now reported for the first time) the case was observed originally in 1878, and has long since been lost sight of, nor was any autopsy record found on examining the pathological records of Kahler's clinique, where it occurred.

S. A. K. WILSON.

**A CASE OF TRAUMA OF THE FOOT OF THE SECOND FRONTAL  
(168) CONVOLUTION, FOLLOWED BY ATAXIA, NYSTAGMUS,  
AND EPILEPSY. F. X. DERCUM, *Journ. Nerv. and Ment. Dis.*,  
Feb. 1905, p. 106.**

DERCUM records the case of a man who was struck on the head. When seen two months later, he presented two scars in the frontal region, the most extensive being on the right side. No evidence of cranial fracture. Intense headache in the region of the scars. Ataxia of the legs, more marked in the left; knee-jerks slightly exaggerated, no ankle-clonus; ataxia and intention tremor of left arm; no affection of face and tongue; no sensory loss. No optic neuritis, but well-marked nystagmus, equal in both eyes; "an irregular atactic movement," elicited only when the eyes were rotated to either side.

During the next year, the general condition, headache, and gait improved; ataxia of the left arm and leg persisted, although in diminished degree; the eye condition remained unchanged; a slight hypæsthesia of the left arm appeared; two attacks of unconsciousness occurred, believed to be of the nature of general epilepsy, not focal—in the first there was no convulsion, in the second "he suddenly rose to his feet, stretched out both of his arms, rolled his eyes . . . moved his head slightly to and fro."

Operation, performed thirteen months after the trauma, disclosed a superficial lesion of the cortex involving especially the posterior portion or foot of the second right frontal convolution.

The head-pain soon disappeared entirely; the affection of the limbs gradually diminished, until, about three years after operation, the ataxia of the legs had quite disappeared, and there was only slight intention tremor and awkwardness in the use of the

left hand; the nystagmus, however, persisted, especially on deviation to the left; two epileptic seizures had occurred.

Attention is drawn to the presence of ataxia, nystagmus, and epilepsy. The combination of the first two symptoms is very suggestive of cerebellar lesion. It is well known that lesions of the frontal lobe may give rise to ataxia. Nystagmus or ocular inco-ordination has not, however, been observed, except in a case recently recorded by Klien—also a superficial traumatic lesion of the foot of the second frontal convolution, with horizontal nystagmus as the special symptom; the two cases are not exactly parallel. The convulsive seizures were general in character, not focal, thus teaching the important practical lesson that “in lesions of the foot of the second frontal convolution, which give rise to epilepsy, the epilepsy is not necessarily focal.” Probably this applies to other portions of the frontal area as well.

A. W. MACKINTOSH.

**CAISSON DISEASE, INCLUDING THE PHYSIOLOGICAL AND**  
(169) **PATHOLOGICAL EFFECTS OF COMPRESSED AIR.** ALFRED  
PARKIN, *Northumberland and Durham Medical Journal*, April  
1905, p. 96.

THE occurrence of cases of compressed air illness, in connection with the construction of the foundations of the High Level Bridge, which is at present being built over the river Tyne, has afforded opportunities for the study of this disease, and facilitated inquiry into its ætiology.

The manifestations of compressed air illness are the same, whether arising from the influence of an increased atmospheric pressure in a caisson, or in a diving-bell, or diving-suit. The cause of the disease, however named, is essentially immersion in an atmosphere of compressed air, and the transit from this to normal atmospheric pressure.

Compressed air is used under a variety of circumstances—in digging the foundations of bridges, in driving tunnels through moist soils, and in mining. The use of compressed air under these conditions is necessitated by the fact that, without such means, a larger quantity of water would enter the chamber from the soil than could be satisfactorily dealt with by pumping. The atmospheric pressure needed, whether in the caisson, diving-bell, or diving-suit, increases in a definite ratio with the depth attained, whether this be in water or in water-bearing soil. In tunnelling, where caissons are used, the same statement holds good. With every 10 metres (or 33 feet) the pressure necessary to keep out water increases one atmosphere, *i.e.* 15 lbs. to the square inch.



At the High Level Bridge works there are three caissons, one at each side of the river, north and south, and one in mid-stream. In structure each caisson consists of a chamber built of steel plates and open below, into which air is pumped, so as to raise the atmospheric pressure in it to the height necessary to prevent the entrance of water. The compressed air-chamber is of such a size as to allow of 34 men working in it at one time. The pressure in the caisson rises from 9 lbs. at the first sinking, to 36 lbs. at the greatest depth attained. The length of the shifts worked varies with the depth and pressure, the shift being shorter at greater depths. Photographs accompanying the essay show the structure of the caissons and air-locks; and diagrams indicate the strata passed through by each caisson in its descent. The nature of the strata affects the ventilation and the constitution of the air in the caisson. In connection with the works, there is a special "medical air-lock" for the treatment of cases of illness by recompression. I have been down one of the caissons, and have recorded my experiences in this essay. In the description of cases, the striking fact is brought out that no ill-effects of any consequence accompany compression. This has an important bearing on the ætiology of caisson disease. Those ill-effects to which the name caisson disease is applied only appear during the process of decompression, or not until some minutes or even hours after it. Pains in the extremities, chiefly in the legs and about the knee, constitute the most frequent symptom of caisson disease. These pains, which are known as "the bends," may be in one limb or in all, both legs and arms. They are of sudden onset, rapidly increasing in severity, and usually, but not always, unaccompanied by any physical sign. Subcutaneous emphysema has been felt by some observers. In one or two cases I have seen continued fibrillar twitchings in the muscles of limbs affected by the pains. With regard to the limbs, there may be temporary paralysis rapidly passing off, while a paraplegia of longer duration, with or without loss of control of the bladder and rectum, is observed in severer cases. Hæmorrhages from the nose and ears are of frequent occurrence. In the worst cases of compressed air illness, unconsciousness of variable duration develops. In those recorded cases which have ended fatally, coma, stertorous breathing, and muscular spasms have preceded death. There have been no fatal cases at these works, but I have observed these serious symptoms in cases which have recovered. I have seen altogether some fifteen cases of compressed air illness. Two were severe cases of paraplegia lasting for several months; the rest were of a less serious nature. Of these latter, eight were cases of "bends" of varying severity. In the remaining five, the prominent symptom was—in one,

vertigo; in three, epistaxis; and in another, deafness. In the descriptions of autopsies on fatal cases, small extravasations of blood into the spinal cord, and softening of the latter, have been recorded; also spaces in the spinal cord filled with round cells, the defined edge of these spaces leading Von Leyden to the belief that they had probably been produced by the escape of bubbles of gas into the nerve tissue of the cord. From my own experiments I have concluded that hæmorrhages into solid organs are the result of the rupture of capillaries by air-bubbles, and not the result of congestion, as the upholders of the so-called "congestive theory" of caisson disease have asserted.

The "air-emboli" theory appears to me to offer the true explanation of compressed air illness; that at a high pressure there is an increased solution of gases in the blood, and when the tension is lowered, *i.e.* in leaving the caisson, these are liberated in the form of bubbles within the blood-vessels and in the fluids of the tissues.

Dr Leonard Hill has recently conducted an interesting series of experiments which throw a great deal of light on this subject, and has embodied his conclusions in a monograph, from which source much of my information has been derived. The circulation is entirely unaffected mechanically by the immersion of the body in compressed air. Dr Hill has actually demonstrated microscopically in the web of the frog's foot, and in the bat's wing, the formation of air-bubbles during decompression. He has also shown that recompression causes the gas to re-dissolve in the blood, and that if, after compression, decompression be slowly effected, no formation of bubbles takes place. By means of a specially constructed compressed air chamber, I have repeated some of the experiments on frogs described by Dr Hill. With the essay are published drawings showing gas-bubbles in various situations in the tissues.

Dr E. H. Snell attaches great importance to free ventilation of caissons, and says that at the Blackwall Tunnel a marked diminution in the number of cases of illness followed an increase in the amount of air pumped into the caissons. On account of the interest which attaches to the disputed question of the value of ventilation as a factor in the ætiology of caisson disease, and with a view to determining, if possible, the part that  $\text{CO}_2$  bears in this latter, a daily record has been kept of the quantity of  $\text{CO}_2$  present in the air of the working chamber of one of the caissons at the High Level Bridge works. The observations are embodied in a chart accompanying this essay. Such data as I am possessed of incline me to the belief that undue importance has been assigned by Dr Snell, and others, to purity of the air of compressed air chambers as essential to the prevention of illness; and this,

too, has, I think, led to a dangerous tendency to minimise the importance of enforcing a slow rate of decompression, which I have come to regard as the one essential mode of preventing the development of illness. Yet, although for the present the value of CO<sub>2</sub> as an ætiological factor cannot be definitely settled, it must be admitted that whatever the gas present in excess in the blood while under compression, the liberation of this as bubbles during decompression can be entirely prevented by enforcing a sufficiently long period for this process.

The prognosis in the majority of cases of compressed air illness is good. The pains pass off, as a rule, spontaneously, usually in a few hours, but may last for weeks. The paralysis is sometimes recovered from even in a few hours. In more severe cases, evidences of permanent cord injury persist in the form of spasticity and ataxia of the legs, and imperfect control over the bladder. In cases of paraplegia with recovery, the cause is probably pressure of air-bubbles, either in or outside the vessels, upon nerve elements of the cord, with no laceration or hæmorrhage. In cases of permanent paraplegia, there has probably been softening, as a result of air-embolism, or rupture of the capillaries by air-bubbles, followed by hæmorrhage.

In fatal cases of compressed air illness, death may be immediate from coma, or may follow later from cystitis or bed-sores. As regards treatment, the pains are slightly benefited by rubbing, but are often so severe as to require morphia. Recompression and gradual decompression form the most efficient means of treating those affected with caisson disease, but must be resorted to early if they are to be of any use. This is carried out in a special "medical air-lock."

Slow decompression is by far the most important means we can employ to prevent the disease. In caisson work the "locking-out" process is almost invariably too short. Divers, also, are too rapidly pulled up, and so decompressed in many instances. The "locking-in" process is of less importance than "locking-out," and, with healthy Eustachian tubes, may be made very short.

In compressed air work generally, there is great need for larger and more comfortable air-locks, so that decompression can be prolonged to the necessary time without being too irksome to the workman. Also it would be of great assistance if the medical air-lock, for recompression in cases of illness, could be more commodious; and this is particularly necessitated for the treatment of the severe cases, when, as may happen, the patient is unconscious, and needs to be in the prone posture with someone at hand to attend to him.

By the observance of proper precautions the dangers of working under a high atmospheric pressure may be reduced to a minimum;

and further, I believe that, by the choice of suitable men, and the proper regulation of the length of shift and time given to decompression, illness can be avoided, and this at greater depths than at the present time reached.

AUTHOR'S ABSTRACT.

# **HYSTERICAL SEIZURES RESEMBLING CORTICAL EPILEPSY.**

(170) (Über den rindenepileptischen ähnliche Krämpfe hysterischer Natur.) WOLTÄR, *Prager med. Wochenschrift*, xxix., No. 52, 1904, S. 673.

THE author reports two cases of what he believes was hysteria, presenting a resemblance to typical Jacksonian epilepsy of the closest, and rendering a differential diagnosis delicate. A servant girl of 17 was the subject of periodic attacks, unassociated, usually, with loss of consciousness, which began by palpitation and an epigastric sensation. These were followed immediately by twitching of the left middle finger, which then fully flexed, and was succeeded by tight closure of the fist; the left arm then twitched in its turn, and the left leg—sometimes the seizure continued on the right side, involving arm, then leg. Headache was a common sequela. Patient discovered herself how to abort the attack by firmly grasping the left middle finger whenever it commenced to twitch. The average duration was from two to five minutes. There was no exhaustion paralysis on the left side after the seizure, for though the arm hung limp, on encouragement the girl could perform any movement well. Systematic examination failed to disclose any hysterical stigmata, as far as the body was concerned, or any evidence of organic disease of the central nervous system. Psychical signs of functional impairment were not wanting, however; several times the attacks occurred during the physician's visit: once when an epileptic fit occurred in another occupant of the ward; and the fact that she was subject to visual hallucinations, and peculiarly amenable to the influence of suggestion, are further points in favour of the diagnosis.

The second case was a more confirmed hysteric, with frequent right-sided clonic movements of the limbs occurring during fits in which there was no loss of consciousness.

The difficulty of the cases is admitted, and various others, more or less analogous, are cited from the literature.

An attempt is made at a differential diagnosis between the clonus of true Jacksonian epilepsy and of hysterical unilateral fits, the movements in the latter partaking more of the nature of rhythmical spasms.

S. A. K. WILSON.

**A CASE OF HYSTERICAL "WANDER-IMPULSE."** (*Wandertrieb* (171) *bei einer Hysterischen.*) WOLTÄR, *Prager med. Wochenschrift*, xxix., No. 44, 1904, p. 565.

THE case concerns a young girl of 17, in whom typical hysterical seizures supervened on a severe head injury. Her previous rather featureless character became profoundly modified by unusual susceptibility to suggestive influence. The first opportunity moulded her whole being in a certain direction. Seduced and abandoned by a false lover, she was seized with the one desire to recover him again, and as he had frequently spoken of making his way to America, the aim of her life was to reach, by whatever means, some port whence she could sail over sea. That the patient was an hysteric is evident enough from the examination made of her condition when under observation, but the view that the impulse to wander which she betrayed was pathological and analogous to post-epileptic phenomena is not so obvious, the circumstances of the case being capable of a simpler interpretation.

S. A. K. WILSON.

**FURTHER CONTRIBUTION TO THE SUBJECT OF MICRO-**  
(172) **GRAPHIA.** (*Weiterer Beitrag zur Lehre von der Mikro-*  
*graphie.*) A. PICK, *Wien. klin. Wchschr.*, Jan. 5, 1905,  
p. 7.

A YOUNG woman, subject to hysterical attacks of the most varied kind, was found on examination during one of them to have a definite degree of macropsia; that is to say, she failed to recognise the familiar figure of the physician attending her, "because he looked so big"; the nurse similarly, "because the nurse she knew was smaller and slenderer." A gulden offered her "was bigger than a gulden"; a crown piece "could not be a crown piece, a crown piece was smaller," etc. During the same time she wrote a letter in which the handwriting was extraordinarily small; some of her ordinary handwriting shown to her was not avowed to be hers; "she never wrote so big as that." The point of interest in the case is the connection, if there is one, between the micrography and the macropsia. It is possible that the latter is the cause of the former, and that a correction by muscular movement of the mistakes entailed by the macropsia occurs, involving for the possibility of its occurrence a normal functioning of what we call collectively the muscle sense. Of interest in this connection is the remark of Köster, that the child who is learning to copy the letters of the alphabet from the school-room card, is eventually able, from the kinæsthetic impressions of hand movements, to

correct the false idea; due to distance, which the retinal image has given him of the size of the letters he has to copy. In a case cited by Ireland, the micropsic illusion disappeared whenever the patient moved his hand towards the objects that seemed so far off. Veragath mentions an instance of micropsia where, during the persistence of the phenomenon, each and every movement of the hand, the arm, the leg, the body seemed to the patient to have assumed enormous dimensions, and argues that interference with the muscle sense, not of intrinsic eye, but of skeletal muscles, is the cause of the micropsia, the centripetal impression conveying the idea of a much bigger muscular movement than the actual one. The author's case may therefore be explained on the view that, owing to the kinæsthetic disturbance, limited movements both of intrinsic eye and of hand muscles appear remarkably extensive, so that, misled by these impressions, the patient makes every movement in writing correspondingly small.

S. A. K. WILSON.

### PSYCHIATRY.

**ON HALLUCINATIONS IN PATHOLOGICALLY ALTERED SENSORY MECHANISMS.** (Ueber Halluzinationen in pathologisch veränderten sensorischen Mechanismen.) A. PICK, *Wien. klin. Wchnschr.*, Feb. 16, 1905, p. 161.

THIS is a contribution to the question, in what way does the previous alteration of a sense organ or its cortical centre influence the hallucinations of that sense? In 1883 Pick called attention to an old observation of Holland, that of an elderly man who after a trauma developed a transitory sensory aphasia: during his first drive on recovery he had auditory hallucinations of paraphasic content. Pick's interpretation was that these hallucinations were the reaction to the stimulus of the drive of a speech mechanism which had not completely recovered. In 1892 he published the case of a general paralytic with a right-sided epileptiform attack, who after the attack had paraphasic auditory hallucinations in the right ear. The present contribution is an observation closely analogous to that of Holland. The patient was a general paralytic forty years of age, who had an attack of paraphasia lasting less than one day. On the day after this attack he complained of hearing in his right ear, and as if coming from the right side, frequent repetitions of things said by other patients. While being wheeled along in a chair he complained that he heard on his right side harsh, senseless syllables; in the evening he had the hallucinations of repetitions, but no longer the paraphasic hal-

lucinations. The hallucinations ceased completely one week after the attack of paraphasia. Special examination of the ear showed normal local conditions.

Pick lays emphasis on the fact that patient turned his head in the direction of the hallucinations, as showing the sensory-motor importance of the auditory speech-centre; the case demonstrates the possibility of unilateral auditory hallucinations of cortical origin.

C. MACFIE CAMPBELL.

**ON DEATH IN FUNCTIONAL PSYCHOSES.** (Ueber Todesfälle bei (174) funktionellen Psychosen.) M. REICHARDT (of Würzburg), *Centralbl. f. Nervenh. u. Psych.*, Jan. 1, 1905, p. 1.

It is usually difficult to determine in a case of a so-called functional psychosis with fatal termination, whether the cause of death be the brain-process underlying the mental disorder, or various conditions such as refusal of food, infection, exhaustion, resulting from the psychosis. The author gives briefly the cases of ten patients presenting the clinical picture of a functional psychosis, usually an acute delirium or catatonia, who died without any sufficient cause save the psychosis itself. He then reports the cases of three patients with a functional psychosis, who before death presented symptoms of organic brain disease, symptoms of chronic compression in one case and choked disc in another. In the two last-mentioned cases there was found to be a great disproportion between skull-capacity and weight of brain; the brain was swollen and congested, but firm, and showing neither oedema nor inflammatory exudate. Reichardt holds that the death in these cases must be referred to the compression of the brain due to its swelling, this latter being caused by the unknown pathological change in the brain which was at the bottom of the psychosis; the deaths in his first series of cases may be due to a similar process of great intensity, but not causing congestion of the brain. He lays stress on the examination of the optic disc, and of the relation of skull-capacity to weight of brain.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO THE STUDY OF DEMENTIA PRÆCOX.** (175) (Contributo allo studio della demenza precoce.) P. GONZALES, *Riv. Sperim. di Fren.*, 1904, p. 765.

THE author gives the following clinical account of a case of dementia præcox of catatonic type:—

The patient was a married woman, aged 26, employed in rubber works, and was admitted to the Asylum in March 1900.

Her father died at the age of 58 of pneumonia, and had been a heavy drinker. Her mother died at 65 of cancer of the stomach. The family history disclosed nothing else worthy of note.

The patient's childhood was uneventful. Menstruation occurred at the age of 17, and was normal in character. At school she showed quick intelligence and lively disposition, and when, after successfully completing her school education, she entered the rubber works, she was rapidly entrusted with delicate and difficult tasks.

At the age of 24 she married. A few months later she had an abortion, but a second pregnancy ended in the birth of a full-time child. Her married life appears to have been free from domestic or pecuniary trouble.

The first signs of mental disturbance were noticed by her husband and relatives exactly six months after the birth of her child. Without any apparent reason she became self-absorbed and slightly melancholic, apathetic and indifferent to her surroundings. She lost interest in her husband and child. Her companions noticed from day to day that her former vivacity was dying out, and that its place was being taken by a sort of apathy and indifference, often characterised by absurd behaviour and strange acts which were sometimes of a violent and impulsive nature.

These phenomena became progressively more prominent. Persistent dislike of food was succeeded by an extreme voracity, along with which came loss of physical condition, sleeplessness, sudden bursts of laughter and tears without any apparent cause. Longer residence in her family becoming impossible, the patient was removed to the asylum.

On admission, her bodily state was weak and presented a marked degree of anæmia. Physical examination of the head, trunk, etc., showed very little worthy of note, and the same may be said of the examination of the various systems—circulatory, respiratory, nervous, etc., with this exception, that she suffered from an intractable gastritis.

For some days after admission she remained in a state of profound depression, with dazed expression and listless manner, much delayed perception and poor ideation. She slept very little, sitting for hours in her bed, immobile, staring in front of her with arms hanging by her side.

After a few days, a period of excitement suddenly supervened, characterised specially by a stupid gaiety, not determined by any appreciable cause. Her memory appeared intact; she was fully cognisant of her surroundings, and if questioned about her former employment could give accurate details of her work. In this period of excitement, sensory reactions showed an appreciable accentuation.



For several months the patient remained in a condition of alternating states of depression and semi-stupor, and of excitement and *bien-être*, the latter always characterised by an increase in hallucinatory phenomena.

With the progress of time, the different symptoms associated with either state became aggravated; in the one she became dirty and resistive; in the other the psycho-motor agitation became more intense and impulsive.

At the end of eighteen months' residence in the asylum the diagnosis of dementia præcox was made, the patient at that time exhibiting marked catatonic symptoms, with progressive physical deterioration. The symptom of "Negativism," as illustrated in the following examples, was prominent: if the patient was asked to fasten her dress, she at once did her best to take off all her clothing; if asked not to spit on the ground, she immediately began to spit all around her, on the seats and walls.

The removal of a finger for caries of the bone was well borne under an anæsthetic; no subsequent change was noted in the mental condition.

Ordinary sensation to touch, pain, heat and electricity appeared to be completely abolished. None of the customary stimuli produced any reaction, even though pushed to a degree which in ordinary individuals would have produced intense pain. In the last months of her life, the evidences of any mental activity gradually disappeared. Severe intestinal symptoms supervened, with rapid emaciation and loss of strength. To the end, however, the patient preserved her resistiveness and rigidity to such a degree that it was possible to lift her from her bed by an arm, with the legs and head and trunk preserving the position they had occupied when she lay in bed.

She died two years and six months after admission to the asylum, with symptoms of chronic enteritis and advanced marasmus.

T. C. MACKENZIE.

## TREATMENT.

**THE TREATMENT OF PUERPERAL ECLAMPSIA WITH PARATHYROIDIN.** (Il trattamento dell' eclampsia gravidica con la paratiroidina.) Prof. G. VASSALE, *Bollettino della Società medico-chirurgica di Modena*, Anno viii., 1904-1905.

In 1896 Vassale and Generali demonstrated that the parathyroid and thyroid glands have independent functions. They showed that the parathyroids, in virtue of an internal secretion, have an extremely important, chiefly antitoxic, action, and that fatal tetany

follows the abolition of their functional activity. The thyroid, on the other hand, has a trophic function in relation to general nutrition. At the outset of this paper, Vassale insists upon the fact that, if some animals escape the fatal and more or less acute consequences of parathyroidectomy, it is because they possess supernumerary or aberrant parathyroids. This conclusion has already a solid basis in certain results of the recorded experimental investigations, but he cites in support of it an interesting observation made by Piana and recorded by Stazzi, showing how impossible it is in some animals to perform a complete parathyroidectomy. Piana had occasion to make sections of the plexiform ganglion of the vagus of a dog, and found imbedded in it a nodule having the structure of the parathyroid gland. Vassale examined the preparations, and was able to confirm Piana's conclusion as to the nature of the tissue.

Partial parathyroidectomy, as a rule, gives rise only to slight and transient tetany, sometimes to chronic recurrent tetanic symptoms. This partial removal is not followed by hypertrophy of the remaining portions of gland tissue. Though the animal may remain in good health there is a latent parathyroid insufficiency, and the characteristic convulsive phenomena may be determined suddenly by various conditions, such as pregnancy, parturition, lactation, chronic eczema, exposure to cold, etc. Among these conditions pregnancy and lactation have been found to have chief importance.

Vassale had under observation for five years a bitch from which, in 1896, he removed all the parathyroid glands, excepting one. Some slight transient tetanic symptoms followed, but thereafter the animal remained well, excepting on two occasions, one during lactation and the other during pregnancy, when it was seized with violent epileptiform convulsions. On both occasions the animal's life seemed to be saved by the administration of large doses of thyroid gland.

The resemblance presented by the symptoms in this case to those described by Weis and Meinert as occurring in women from whom goitres had been partially removed, led Vassale, in 1901, to express the opinion that all forms of tetany associated with maternity were due to functional insufficiency of the parathyroids. More recently, Nicholson and Baldovsky have recorded success in the treatment of puerperal eclampsia by large doses of thyroid gland. Vassale contends that in these cases, as in the experimental observation just referred to, the beneficial effects were due to the action of parathyroid substance contained in the thyroid glands, the internal parathyroids being embedded in these organs. He has succeeded in extracting from the parathyroid glands of the ox an active substance to which he has

given the name of parathyroidin or parathyroid antitoxin. He has demonstrated its efficacy by subcutaneous injection, as well as by internal administration, in dogs after performance of parathyroidectomy. In the meantime the preparation of this substance has been entrusted to the Institute of Serum-Therapeutics at Milan. It has already been tried in three cases of puerperal eclampsia with immediate and complete success.

Whilst there is experimental evidence that pregnancy is of itself capable of rendering active a latent parathyroid insufficiency, there are grounds for believing that exogenous and endogenous intoxications, by causing a greater call to be made upon the antitoxic functions of the parathyroid glands, may aid in bringing about this result. Experimental and histological investigations seem to prove that the cells of these glands are of the nature of those tissues which, owing to extreme differentiation, are in the adult incapable of regeneration. If, therefore, they are injured, or happen to be congenitally deficient, they cannot undergo hyperplasia, and the individual so handicapped will be liable to the sudden onset of symptoms of parathyroid insufficiency when, owing to certain forms of exogenous and endogenous intoxication, an unusual strain is put upon the parathyroid function. There are now many recorded observations which show that intercurrent toxæmias may determine the onset of convulsions in animals upon which partial parathyroidectomy has been performed. It is therefore easy to understand how, in the pregnant woman, renal or hepatic insufficiency, bacterial infections, intoxications of placental or foetal origin, etc., added to the already increased call made upon the parathyroid function by pregnancy, may, if there is an insufficiency of this function, determine the sudden onset of convulsions.

Parathyroidin has also been used with successful results in a case of tetany in a child of two and a half years, and the author discusses the question of the possibility of some forms of convulsive disorders in children being amenable to treatment by this new therapeutic agent.

He has also tried parathyroidin in three cases of ordinary epilepsy, with markedly beneficial results in two of them, but he recognises the necessity of making further observations before forming any judgment regarding its therapeutic value in this disease. Its effect in exophthalmic goitre has yet to be made the subject of investigation. Vassale considers that certain of the phenomena of this disease result from the involvement of the parathyroids in the morbid process that affects the thyroid, and that, therefore, administration of parathyroidin may be found to be beneficial.

Lastly, in view of the very severe character of the disturbances

of the higher cerebral functions caused in dogs by parathyroidectomy, he urges that parathyroidin should be tried in various forms of insanity. He thinks it is not improbable that in many cases the improvement resulting from thyroid treatment may be due to the small quantity of parathyroid substance contained in the thyroid gland.

W. FORD ROBERTSON.

**ON THE QUESTION OF THE JUSTIFICATION OF THE ARTIFICIAL INTERRUPTION OF PREGNANCY FOR THE PURPOSE OF CURING PSYCHOSES.** (Zur Frage nach der Berechtigung künstlicher Unterbrechung der Gravidität behufs Heilung von Psychosen.) A. PICK, *Wien. med. Wchnschr.*, No. 2, 1905.

THE author, after reviewing the opinions of various authorities, still considers that the above question is not decided. He points out the need of a renewed study of the whole subject by the aid of modern pathological methods. The chief argument that has always been raised against the induction of abortion in these cases is that delivery at full time is not followed by much improvement. Pick dissents from this view, and lays stress on the fact that there are many cases on record in which the improvement following upon abortion has been most marked—often indeed of the nature of a cure.

Whatever interpretation one chooses to put upon the factors responsible for the psychoses of pregnancy, it must be granted that their long and continuous existence must exert a harmful influence on the altered brain conditions underlying the psychoses. Therefore the correction of these factors, or, it may be, their removal, at the right time, may reasonably be expected to improve the condition of the nervous system. Hence Pick strongly advocates that a large proportion of such cases should be treated by the artificial interruption of gestation. At the same time he recognises that there is difficulty in selecting those cases which are most suitable for this treatment, and much discrimination is needed in deciding this matter.

OLIPHANT NICHOLSON.

**A REPORT ON THE CARE OF THE INSANE POOR.** By a SPECIAL COMMISSIONER of the *British Medical Journal*, *Brit. Med. Journ.*, Jan. 7 to March 18, 1905.

THE subject with which this series of eleven articles deals is of great importance from the administrative point of view. In the introduction the Commissioner draws attention to the serious over-

crowding in the County and Borough asylums of England and Wales which the last Annual Report of the English Commissioners in Lunacy reveals, and to the discomfort, retarded recovery rate, increased death-rate from asylum dysentery and tuberculous diseases which result from the herding together of so many diversely affected patients, and also to the impossibility, under present conditions, of giving that individual attention to patients which is essential for their proper treatment. He then proceeds to an examination of the systems of the family care of chronic, inoffensive lunatics as practised in Belgium, Scotland, France, Germany and Holland, with particular reference to the feasibility of its adoption in England and Wales (*a*) as a means of disburdening the asylums of a considerable proportion of their harmless, incurable cases, and so bringing about a concentration of medical effort on the acute and curable cases; (*b*) as a more humane method of treatment, by substituting natural domestic influences for an institutional environment, the latest development, in fact, of the policy of the "open door"; (*c*) as a means of considerably lightening the burden which the care and maintenance of the insane throw upon the community; (*d*) as a means of testing the fitness of a convalescent for a return to society; and (*e*) as a therapeutic measure in itself.

The Commissioner visited the colonies of Gheel and Lierneux in Belgium, Dun-sur-Auron and Ainay-le-Chateau in France, Uchtspringe, Gardelegen, Jerichow and Rockwinkel in Germany, Veldwijk in Holland, and one or two centres in Scotland, and gives the impressions he received and also analyses of the statistics of the various colonies. His impression was throughout favourable, and the statistics show a remarkable drop in the death-rate as compared with closed asylums, particularly with regard to tuberculous diseases and asylum dysentery—the latter practically disappearing as a cause of death—and also a steady appearance of recoveries amongst cases previously considered incurable, and which had for many years—in some cases for as many as twenty years—been confined within asylums. The Commissioner was struck with the improved physical condition of the patients, and the still more striking amelioration in their mental condition, patients formerly agitated and restless becoming tranquil under domestic influences and a natural occupation; cases with stable delusions of persecution improving as a direct consequence of the removal of a confinement which sharpened their resentment against imaginary persecutors; and cases which had been actively suicidal within the asylum ceasing to make any attempt on their lives. The possibility of dangerous occurrences is dealt with in the account of each colony, the methods of inspection and medical care are described and the conclusion drawn

that, under adequate inspection and control, the possibility of dangerous occurrences to the public and to the patients themselves may be entirely eliminated. Perhaps one of the most significant parts of this report is contained in the "Conclusion," in which the boarding-out system in vogue in England and Wales is shortly dealt with. From this it appears that no less than 5516 pauper lunatics were living under family care in England and Wales in 1903. These patients are boarded out with their relatives or friends, generally their parents—that is, with "the very people whom universal experience has proved to constitute the worst kind of guardian"—they are detached from the general lunacy administration of the country, are under the control of the Board of Guardians and drop out of the supervision of the Commissioners in Lunacy, and are visited but once a quarter by the parochial medical officer, who is required to fill in a report as to their bodily condition, maintenance, etc., and (the italics are the reporter's) *information as to the employment of mechanical restraint*. The Commissioner sums up the results of his study in the following terms:—

A. *The patients are happier, healthier and more tranquil than in the asylum.*

B. *The application of this method results in a large pecuniary benefit to the tax-payer: (a) by obviating the necessity for erecting new asylums or extending existing structures: (b) by the intrinsic cheapness of this form of provision for the insane: and (c) by the aid in money and labour afforded to their guardians.*

C. *The system is one of increased, not diminished, co-ordination and control; and*

D. *The application of family care has a beneficial influence on the treatment of the insane, both in the asylum and in those under family care.* The whole subject is ably reviewed in a leading article in the *British Medical Journal* of March 18, 1905, and it is to be expected that the work of the *British Medical Journal* in this field will be instrumental in removing the groundless fears and prejudices with which this system is still regarded by many, and in forwarding much needed reforms in lunacy administration.

R. CUNYNGHAM BROWN.

## Obituary

### SIR JOHN SIBBALD, M.D., F.R.C.P.E.

FEW alienists or specialists mingled so much in the general interests of the profession as, during his long and active life, did Sir John Sibbald; and probably few of them formed so many friends and were so popular with members of all branches of the profession as he was. His early training in medicine was so wide that he never afterwards lost either touch with, or interest in, the general progress of medicine. That aloofness from pure medicine which is the blot of specialism could not be urged against him, and throughout a long official career he never lost sight of the fact that he was before everything else a physician. To these facts his success was largely due, and they also account in great measure for the regard in which he was held by his brethren of the medical profession.

Born at Edinburgh in June 1833, Sir John was within a few months of completing his seventy-second year when he died on the 20th April 1905. He was educated at Merchiston Castle School and at the University of Edinburgh, where he graduated as Doctor of Medicine in 1854. After graduating he became House-Surgeon to the Perth Infirmary, from whence he proceeded to Paris to complete his medical studies. His associations with the Paris Medical School were among the most pleasant of his life, and he always maintained cordial relations with successive generations of leading doctors in that city. From the Paris Schools he came to London, and after walking the hospitals for some time he became Resident Physician at the Brompton Hospital for Consumption. About this time he appears to have conceived a predilection for the study of nervous and mental diseases, which led him to return to Edinburgh and to become Assistant-Physician at the Royal Edinburgh Asylum, Morningside, under the late Dr Skae. The care of the insane in Scotland at that time, to a young, progressive mind like Sibbald's, left much to be desired. On a salary, as he used often to say, of only sixty pounds a year, he made several pilgrimages to the Continent, and studied there whatever he found most useful and commendable. He contributed his impressions freely to the British journals, and his article on Gheel, written in the *Journal of Mental Science* in 1861, did more than anything

else to direct the attention of people in this country to the advantages of the colony system in the care of the insane. At this time also he formed a lasting friendship with Professor Griesinger of Berlin, and it is safe to say that the influence of that master mind moulded the after-course of his professional and official life. Their temperaments were kindred, and Sibbald easily assimilated the progressive ideas of his friend. In 1862 he was appointed Medical Superintendent of the Argyle and Bute Asylum, where he had full opportunity of putting into force those changes in care and treatment which he regarded as beneficial. In 1870 he was appointed a Deputy Commissioner in Lunacy, which appointment necessitated his retiral from medical work and his resignation of the Editorship of the *Journal of Mental Science*, which he had held for a short time previously. In 1877 he delivered the Morison Lectures before the Royal College of Physicians, Edinburgh, choosing as his subject, "Insanity in its Public Aspect." In 1878 he was appointed a Commissioner in Lunacy. From the latter office he retired, under the age limit, in 1899. During the six years which have elapsed since his retirement from office he never ceased from the labour of promoting by every means in his power the interests of the speciality and the good of the insane. His small work entitled "Plans of Modern Asylums," published in 1897, led to his accepting the post of medical adviser to the Edinburgh District Lunacy Board in the work of selecting the plans for the construction of the new asylum at Bangour.

He interested himself in various philanthropic and scientific pursuits, including the organisation of Congresses for the international study of various forms of home relief, and he was chairman of the executive committee of the Congress for this purpose so successfully held in Edinburgh last year. His paper on the statistics of suicides in Scotland, read before the Royal Society of Edinburgh in 1900, was the outcome of years of study, and received on all hands the consideration which it deserved. As a writer he was precise, scrupulously exact in detail, and guarded in the expression of opinion. In his daily life he was cautious, persuasive, and, on account of his sound judgment, influential. To those who knew him intimately he displayed a character of great ethical beauty, the prominent qualities of which were a strict adherence to principles without offensiveness, geniality, and generosity.

The last months of his life were clouded for his friends by the sad and hopeless nature of the malady which carried him off. He himself was the least concerned of all, meeting the coming change with a fortitude which those who were privileged to witness it can never forget.



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THE FIFTEENTH CONGRESS OF FRENCH ALIENISTS AND  
NEUROLOGISTS.

WE have been asked to announce that the above Congress will be held this year at Rennes from the 1st to the 7th of August, under the presidency of Dr A. Giraud, Director of the Asylum of Saint-Yon (Seine-Inferieur).

The Congress is open to physicians of all nationalities.

The subjects chosen for discussion are :—

1. Psychiatry.—Hypochondria, introduced by Dr Roy of Paris.
2. Neurology.—Ascending Neuritis, by Dr Sicard of Paris.
3. Treatment.—Balneology and Hydrotherapy in the treatment of mental affections, by Dr Pailhas.

An important place is reserved for original communications on psychiatric and neurological subjects, and for the presentation of patients and specimens.

Further information regarding the Congress may be had from the Secretary, M. le Dr J. Sizaret, Rennes.

# Review of Neurology and Psychiatry

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## Original Articles

### ON FOUR FIXED VERTEBRAL POINTS AND THE VARIATIONS IN THE SUBJACENT SPINAL SEG- MENTS IN TWENTY-TWO AUTOPSIES.

By Dr L. J. J. MUSKENS, Amsterdam.

THE progress made during the last fifteen years in the local diagnosis and the operability of new formations of the spinal cord, has made the problem of a more definite localisation of the different spinal segments in their relation to the vertebral column one of practical importance. Sir William Gowers, in his text-book, was the first to publish details in this respect. Sir Victor Horsley has from the beginning called attention to the fact that the point of greatest compression is usually situated far lower than would be expected from physiological data—a fact which must be partially ascribed to the disproportion between length of cord and spine.

The only special papers I have found regarding the subject are those of Reid<sup>1</sup> and Chipault.<sup>2</sup> The former took accurate notes from six individuals. His method was to lay the body horizontally, and place a sheet of glass upon it; a drawing was then made of the spines, and later on, after the cord had been laid bare, of the cord and its roots. Later, Allen Starr and Bruns have given more details about the disproportion in length between the cord and the vertebral column.

Pure anatomists had little interest in this matter. They

<sup>1</sup> *Journ. of Anat. and Physiol.*, vol. xxiii., 1889, p. 343.

<sup>2</sup> A. Chipault. *État actuel de la Chirurgie nerveuse*, 1902, Forme i.



observed that the outgrowing spinal roots, especially those of the lumbar region, remain a long way within the spinal canal after their exit from the cord, but they do not give numbers and exact localisations. The interest of comparative anatomists was directed especially to the shortening which the spinal cord as well as the vertebral column underwent, both at their distal and proximal ends. Rosenberg in particular has shown that in the present higher vertebrates both the column and the cord are undergoing a shortening, the result of which is that in man and in the anthropoid apes individuals are found possessing two or three vertebræ more than the average, and on the other hand, some individuals with specially short vertebral columns possess a smaller number of vertebræ. At the same time, although not going parallel with the said process, a shortening of the spinal cord takes place, as well in absolute length as in the number of segments. Both shortening processes affect as well the upper as the lower end. The shortening of the cord advances more rapidly than that of the vertebral column. As the result of this, the fact has to be considered that, according to Bolk and others, there are many cases in which no trace is found of the first cervical spinal nerve, at least of the motor root, the posterior root being constantly present, in part at least, in all individuals.

Further reference to these questions is not necessary; I shall merely state here the results obtained from 22 autopsies. After a careful search as to the possibility of using other landmarks than the spinal processes, Chipault failed to find any of practical use. Moreover, Gowers, Reid, and Allen Starr came to the same conclusion, so that I did not hesitate to use these again as fixed points. In variance with the former workers on the subject, it appeared to me necessary to extend the number of bodies examined as much as possible. For in spinal surgery it is a matter of prime importance, as well for the success of an operation as for the subsequent wellbeing of the patient, that the number of spinal arches removed be as restricted as possible. No means, therefore, are to be regarded as superfluous which enable us to locate as precisely as possible the limits between which the point of greatest compression might be found in extreme cases—even in the case where a spinal cord of a prefixed type, *i.e.* one with an unusually high position, should coincide with a vertebral column which is extremely short, as well in absolute length as in the number of

vertebræ. It is only if he has carefully considered all these possibilities, and has taken into his calculation also the improbable chances, that the surgeon can be regarded as having discharged his duty. It is clear that such a calculation can only be based on data obtained from a great number of bodies.

In every case the procedure which I followed was to fix four spinal processes and to note, after laying bare the cord over its whole length, which spinal segments underlay these fixed points. The spinal processes selected were the 4th cervical, 1st dorsal, 7th dorsal, and 12th dorsal. The body was laid horizontally, face downwards; long nails were fixed into the vertebral column exactly at the level of the mid-point of the above-mentioned spinous processes, but one inch to the side, in order that the exposure of the cord should in no way be interfered with.

It is not infrequently very difficult to locate exactly the chosen fixed points, viz., the spines of C 4, D 1, D 7, and D 12, especially C 4. Because the prominent spine (*vertebra prominens*) appeared to belong sometimes to the 7th cervical vertebra, at other times to the 1st dorsal vertebra, it was not possible to make use of this landmark. On the other hand, it was not rarely difficult to state exactly which root we had to deal with in a given case. The process of shortening of the spinal cord causes a great variability, especially in the upper part of the cord. In some individuals, as is well known, the first cervical nerve is represented merely by a small anterior and posterior rootlet; in others the motor part of this nerve is entirely wanting. The usual method of locating the different nerve roots, viz., to count the first thin root below the long nerve roots of the cervical plexus as the second dorsal nerve, proved to be very useful in this research. In those rare cases in which the second dorsal nerve contributes to the formation of the brachial plexus, one might make a mistake with regard to this nerve, so that it appeared to be always advisable to control the number of roots from above downwards.

An important detail in this research appeared to be the very frequent difference in the course of the rootlets on the right and left sides. In some cases the same rootlet, for example, which joins the fifth on the left side, helps to constitute the fourth on the right side. It was not infrequently seen that a rootlet leaving the cord amongst the rootlets of the fourth cervical nerve does not

TABLE I.

No.	PSYCHOSES.	AGE.	SEX.	C 4.		D 1.		D 7.		D 12.
				R.	L.	R.	L.	R.	L.	
I.	Dementia paral.	35 years	female	5-6	4-5	3-4	2	9-10		Terminal point.
II.	Dementia paral.	38 years	male	6	6-7	5	4-5	9-10	10-11	Terminal point.
III.	Idiocy	42 years	female	4-5	5-5	3	5-6	10-11	11	1 cm. above point.
IV.	Dementia paral.	40 years	female	4-5		3		10		1 cm. below point.
V.	Dementia senil.	82 years	male	4		1-2	3	9		2 cm. above point or L. 2-3.
VI.	Idiocy	42 years	male	5-6		4-5		10-11		0.5 cm. above point.
VII.	Dementia secundaria	55 years	male	5		3-4				3 mm. above point.
VIII.	Dementia senil.	82 years	female	5-6		2-3	3	8-9		L. 2-3 spinal ganglia are situated free in the vertebral canal.
IX.	Dementia senil.	65 years	female	5-6		2-3	3	8-9	9-10	L. 4.
X.	Idiocy	42 years	female	5		3	4-5	11-12		1 cm. below point.
XI.	?	30 years	female	5-6		3	2-3	9-10	8-9	1.5 cm. below point.
XII.	Idiocy	45 years	female	6		3	2-3	9		1.5 cm. above point.
XIII.	Dementia senil.	68 years	female	5-6		3	3-4	9	8-9	5-4.
XIV.	Dementia paral.	43 years	female	5-6	6	4-5	3-4	8		2-3.
XV.	Epilepsy	45 years	female	4-5	6	3	3-4	9	8-9	Terminal point.
XVI.	Dementia paral.	45 years	male	5-6	6-7	3	3	8-9	10-11	Terminal point.
XVII.	Dementia paral.	48 years	male	5-6	4	3	2-3	6-7		Terminal point.
XVIII.	Dementia paral.	43 years	female	5-6		4		10-11	11-12	0.5 cm. below point.
XIX.	Stupor	45 years	female	4-5		5-6		7-8		L. 2, L. 3-4 cm. above point.
XX.	Idiotia	25 years	female	4-5		C 8		D 8		L. 3.
XXI.	Hydrocephal.	5 years	male	4-5		1-2	2	9	9-10	1 cm. below point.
XXII.	Syph. hered.	4 years	male	4-5		3	2-3	8	8-9	L. 2-3 terminal point across L. 3-4.

join that root, but crosses its lower rootlets and joins the fifth. Occasionally it is seen that single rootlets arising from several roots join a spinal root which leaves the cord at a considerably higher level.

In this way the segmental partition of the cord on the right and left sides may be very different, as will be clear from the tables. In regard to this point, in order to eliminate as much as possible mistakes of this kind, the number of the roots on both sides was determined independently, starting from the second dorsal root. Furthermore, where the nail head lay rather nearer the upper of two roots, between which it passed, the fixed point was held to coincide with the higher or proximal segment.

These facts appear to show a remarkable asymmetry which may be found in the partition of the roots and perhaps also of their grey matter in the human cord.

Another fact which must be kept in mind is that not rarely the cervical part of the cord is found to be of an extraordinary length, whereas the dorsal segments in some cases are of extraordinary small dimensions. Further important differences may be found in the relation of the lumbar part compared with the dorsal and cervical parts. Very often in male cords the lumbar part was found extraordinarily long as compared with that of female and infantile cords.

In order to get a summary I have made a table in which the details are given as much as possible in such a way that, where one of the fixed points fell between the exits of two roots, the fixed point was noted down as belonging to the root below as well as to the root above. In order to get equal results, it was necessary to count all the cases double. If, for instance, on the right side the fourth cervical spine coincided with a point lying somewhat above the middle of the fifth cervical segment, and on the left side a little below the middle of that segment, that is noted down R(ight) = C 4-5 and L(ef) = 5-6. This reduction is necessary in order to get results of practical value.

In order to be able to give a graphic representation of the second table, in which the relative frequency of the males and females is noted down, I have in the many cases which are noted in the first table as C 4-5 and C 5-6 counted them partly with the above, partly with the lower segments.

TABLE II.

	C 4.	D 1.	D 7.	D 12.
Male bodies {	2-C 4 3-C 5 6-C 6 ...	2-D 2 6-D 3 5-D 4 ...	1-D 7 2-D 8 3-D 9 3-D 10	Coincides with point of the cord or 0.3 to 2 cm. above.
Female bodies {	2.5-C 4 7-C 5 4.5-C 6 ...	3-D 2 7-D 3 3-D 4 3-D 5	1-D 7 3-D 8 8-D 9 4-D 10	Coincides with L. 2 in some cases, up to 15 mm. below the lower end of the cord.
Children {	1-C 4 1-C 5 ...	1-D 1 3-D 2 1-D 3	1-D 8 2-D 9 ...	Coincides with L. 2-3 in one, 1 cm. below the lower end of the cord in the other case.

This second table is represented as exactly as possible in Fig. 1, in such a way that the darker stained segments indicate the segments which most frequently coincide with the fixed point of that region. Thus it is apparent that the first dorsal spine coincides once with the 1st dorsal segment, 3.5 times with the 2nd, 8.25 times with the 3rd, 4.25 times with the 4th, 2.25 times with the 5th, and 0.75 times with the 6th dorsal segments.<sup>1</sup> Therefore in the figure the 3rd dorsal segment is stained darkest.

From Table I. we conclude that, as a rule, the cord is shorter in the male; whereas the female type is, on the one hand, more variable, and, on the other hand, reaches lower down than the 12th dorsal spine. It is principally to the female cord that the remarkable fact must be ascribed that, as is visible in Fig. 1, there are two maxima regarding the fixed point, D 12; one is L 5, and the other is S 4. The infantile type has equally a relatively longer cord, and may be classed along with the female.

In order to get a comparable table from Reid's work, I have constructed, from the very explicit and detailed account given by that author, the spinal cords of his six specimens in

<sup>1</sup> This result is the more striking as in none of my individuals were any lordotic or kyphotic conditions found, which should fall outside the range of normal conditions, one child excepted.

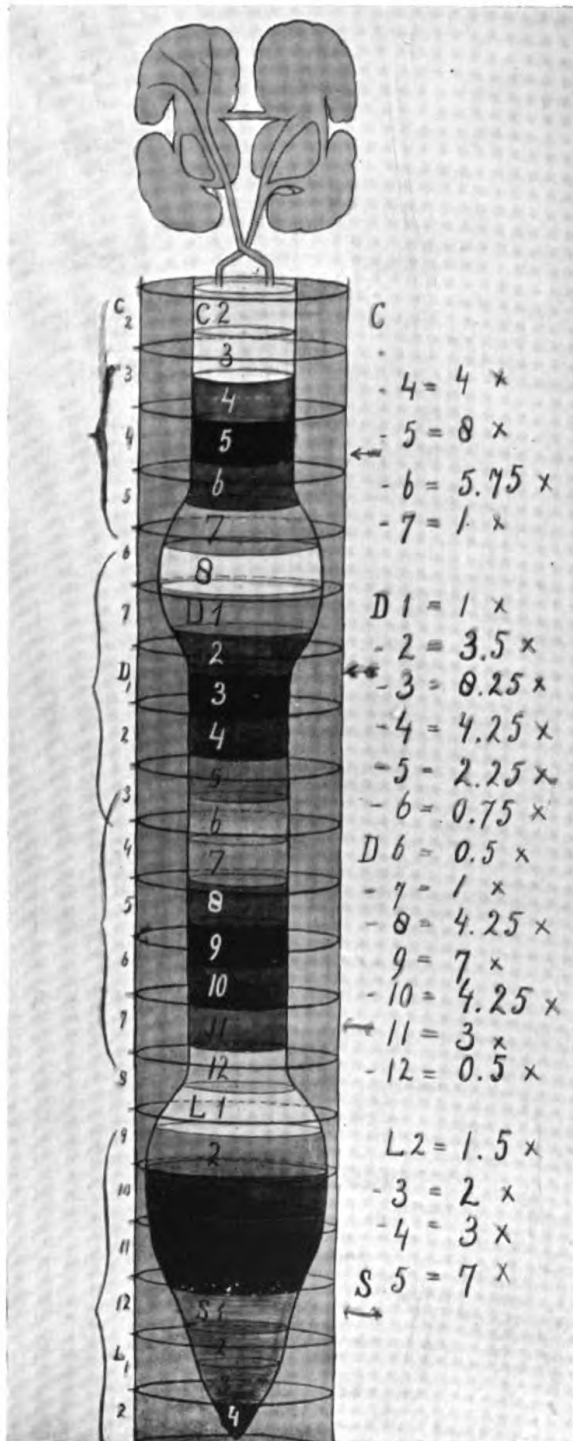


FIG. 1.

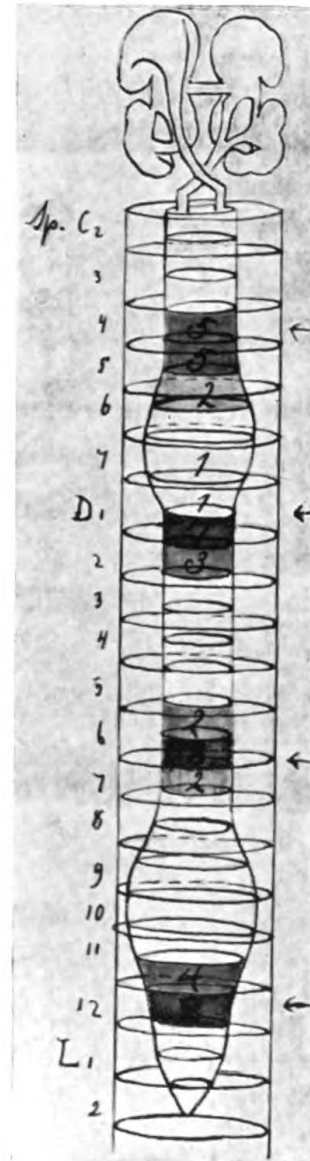


FIG. 2.



their relation to the vertebral column. From these reconstructed drawings I gather the following results :—

TABLE III.

	FIXED POINTS.				
	C 4.	D 1.	D 7.	D 12.	
Case I.	C 5-6	D 3	D 10	S 1	Graphically this Table is represented by Fig. 2, which is com- parable to Fig. 1.
" II.	C 6	D 3-4	D 10-11	S 1	
" III.	C 5	D 3	D 9-10	L 5-S 1	
" IV.	C 6-7	D 3-4	D 10-11	L 5	
" V.	C 6-7	D 3-4	D 10	S 1	
" VI.	C 5	D 1-2	D 9-10	L 5-S 1	

We see, therefore, that the individual variations in Reid's specimens were not so marked as in my cases. This is the more strange, as Reid did not go into any more detailed description (age and sex) of the individuals. Whether the mode of suspending the bodies had anything to do with this discrepancy of our results I cannot say, especially in regard to the difference found for the roots going out from the cord below D 12. I am inclined to think that the difference in method may be largely responsible for this difference of result.

The most useful conclusion from this research is, I think, that in an operative case we have, in the first place, to take into account the immense individual variations, and the possibility, in every case, that we may have to deal with a *pre-* or with a *post-fixed* cord (*i.e.* one in which the segments have a relatively high or a relatively low situation). If these spinal conditions are disregarded, an exploration in a case of compression-myelitis may completely fail, and has, indeed, failed more than once under circumstances otherwise favourable. As a rule, it will be useful to take as landmark for the operation and middle of the incision the spinous process, under which there is most chance of finding the segment that is sought; and, as the uppermost and lowermost points of incision, those spines to which, in extremely *prefixed*, and, on the other hand, in extremely *postfixed* cases, the segment sought for may be subjacent. As a rule, it will be sufficient to remove 2 or 2½ vertebral arches.



The practical use of this Table and of Fig. 1 is as follows :—  
Let us suppose, *e.g.* that the highest point of compression is about the eighth cervical segment. This segment lies at equal distance from segment C 5 and segment D 3. The first is, as a rule, subjacent to spine C 4, the second to spine D 1. Since vertebrae and segments in this region do not usually differ very much in vertical dimension, we expect segment C 8 to be under the sixth cervical spine. Since the Table and Fig. 1 show that in this region the variations downwards are more important than those upwards, we shall therefore further expose three lower and two upper segments, and shall then be practically certain that the spinal region to be explored is accessible for inspection. As a rule it will be possible, after laying bare the two segments which are most probably the seat of disease, to locate the point of compression by probing upwards and downwards.

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### **ATROPHY OF BONE IN PROGRESSIVE MUSCULAR DYSTROPHY.<sup>1</sup>**

By WILLIAM G. SPILLER, M.D.

Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania.

(From the Philadelphia General Hospital.)

IMPLICATION of the bones in progressive muscular dystrophy is not of frequent occurrence, or at least is not often observed, and in many of the text-books on nervous diseases no mention is made of it. The only cases in the literature I have been able to find are those of Le Gendre, Friedreich, Lloyd, Schultze, and Marie and Crouzon. Oppenheim in the fourth edition of his text-book refers to Jendrassik. Marie and Crouzon in 1903 reported a case of muscular dystrophy with fracture and atrophy of bone.

It is probable that the atrophy of the bones is slight in most cases, and therefore is not detected during life. In 1898 I reported a case of progressive muscular dystrophy in which I believed that either bones or joints were diseased. I have recently had another case in which arrest in development

<sup>1</sup> Read at a meeting of the Philadelphia Neurological Society, Feb. 28, 1905.

and probably also atrophy of bone were very pronounced. Scoliosis was present as in the cases of Lloyd, and Marie and Crouzon.

It is not always easy to make the correct diagnosis in cases of muscular atrophy beginning early in life, and a case reported recently by Italo Rossi shows how difficult it may be to distinguish clinically between anterior poliomyelitis and progressive muscular dystrophy.

When the bones are affected in childhood as a result of acute anterior poliomyelitis the condition, as Dejerine remarks, is one of arrest rather than of atrophy, and diminution occurs in the length as well as in the thickness of the bone.

In the case I now report the gradual development of the muscular atrophy, the commencement at the age of two years, and the progression of the atrophy until the age of sixteen years, the partial atrophy of one of the muscles (the right triceps), not uncommon in muscular dystrophy, the implication of the bones and muscles of the face, and the absence of shortening of any of the long bones, are in favour of a diagnosis of progressive muscular dystrophy. Occasionally the muscles of the face may be atrophied as a result of acute anterior poliomyelitis, as Gowers remarks. The asymmetry of the muscular atrophy in the upper limbs in my case is suggestive of acute anterior poliomyelitis, and yet the right upper limb has not escaped, as the triceps muscle is much atrophied. The atrophy of bones in this case has been established by a Röntgen ray examination.

The case can hardly be regarded as one of the hereditary or family type of progressive spinal muscular atrophy of childhood, described by Werdnig, Hoffmann, and Bruns. In this disease death occurs usually in early life. The case I report is as follows :—

J. L., 44 years of age, labourer, is a patient in my service at the Philadelphia General Hospital. He was perfectly well until he was two years of age. At that time he began to get weak, and the muscular atrophy progressed gradually until he was 16 years of age, when it was as intense as it is now. He says he had no acute disease in early childhood.

His face is very much atrophied, and equally so in all parts, although possibly the right cheek is more sunken than the left. The bones of the face also seem to be affected. The atrophy is

intense in all parts of the face, but the orbicularis oris and the orbicularis palpebrarum are not especially involved. He can pucker up his lips and whistle feebly, and can close his eyelids firmly. The tongue is not wasted, and is protruded straight. In showing his teeth he draws up each corner of the mouth fairly well, and can draw up each corner separately and proportionately to the amount of muscle remaining. Sensations for touch and pain are normal in the face. The masseter muscle contracts well on each side, but its power is diminished. The left pupil is a trifle larger than the right. The contractions of the irides to light and in convergence are normal. The extraocular muscles are normal.

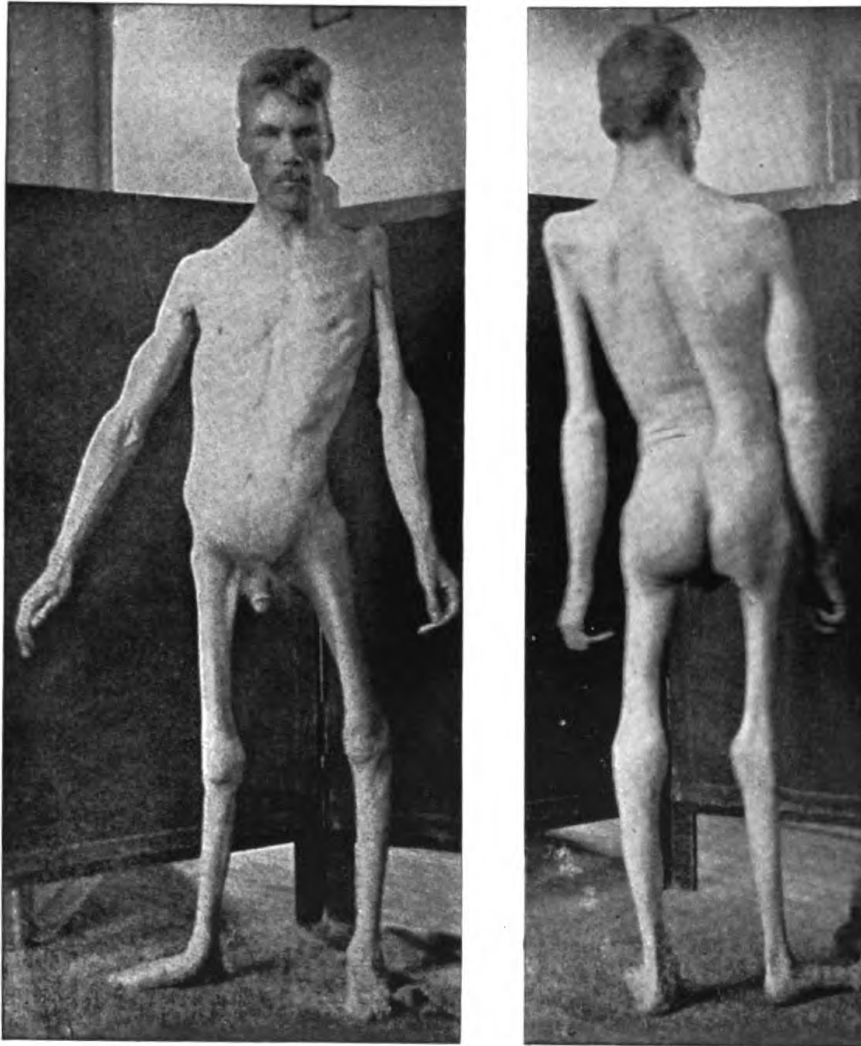
The left upper limb is greatly wasted, especially from the shoulder to the elbow. The left hand is also much wasted. The upper limbs are flaccid. The grasp of each hand is good, that of the right being better than that of the left. The biceps and triceps tendon reflexes are absent in the left upper limb, and there is no muscle here to contract; these reflexes are very feeble in the right upper limb. The right upper limb is fairly well developed, except that the triceps muscle is greatly wasted in its lower part, the upper part of this muscle standing out in a lump. Sensations for touch and pain are normal in the upper limbs.

The muscles of the shoulders and trunk are greatly wasted especially those of the left shoulder.

Both lower limbs are much atrophied in all parts, the right more so than the left. Contractures are not present anywhere. Foot-drop is present on both sides, but the feet can be placed passively in almost normal position. All the toes can be moved voluntarily. The lower limbs are very flaccid. The patellar tendon reflex and Achilles tendon reflex are absent on the right side. The Achilles tendon reflex is very weak on the left side, and the patellar tendon reflex is absent on this side also. Babinski's reflex is not present on either side. Fibrillary tremors are not detected. Sensations of touch and pain are normal in the lower limbs.

Dr M. Kassabian made a Röntgen ray examination and gives the following report:—

“The skiagraph presents a posterior view of the left upper limb, thorax and both shoulders. The humerus and scapula on



DR SPILLER'S CASE.



the left side are smaller than those of the right side, and the epiphyses on the left are united. The left acromion process and glenoid cavity are unusually small, and the head of the humerus appears as if dislocated forward. The humerus seems to be deficient in lime salts. The ribs on the left side are larger than those on the right side.

"The skiagraph of the knees shows simply that the knees are smaller than normal, and probably deficient in lime salts.

"There is no arrest in the development of the bones from injury of the epiphyses."

I am indebted to Dr R. Pemberton for the photographs.

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### A CASE OF GANGLIONIC NEUROGLIOMA.

By LEONARD FINDLAY, M.D.

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So far as I can ascertain, there are very few cases of ganglionic neuroglioma on record, and, on consulting the standard works of pathology, it is found that the authors either dismiss the subject with a very few words, or even doubt the existence of such a condition altogether. Consequently the following case, which came under my notice, and which I consider to be an example of this form of tumour, may not be without interest.

The patient was a male child, æt. 14 months, and was admitted to the Royal Hospital for Sick Children, Glasgow, on 5th October 1903, under the charge of Dr Geo. S. Middleton, during my tenure of office as house-physician in that institution. According to the mother, the patient was a plump, healthy child

when born, and continued to enjoy good health until about three months previous to admission to hospital. About that time he developed a short cough, wheezing in the chest, and shortness of breath. The family history was unimportant.

On admission to hospital the patient was pale and emaciated and slightly rachitic. He had a severe cough, and some expectoration, obtained by swabbing the throat, revealed the presence of tubercle bacilli and diplococci (pneumococci?). The respirations were rapid, numbering 40 per minute, but were not laboured, and examination of the chest revealed a generalised inflammatory condition of both lungs with consolidation in the left lower lobe, and probable excavation in the left upper lobe. The pulse, which was small and regular, numbered 130 per minute. There was no enlargement of the precordial dulness, and the heart's sounds were pure. The tongue was slightly coated but moist. The abdomen was not distended, nor was there any tenderness on palpation. There was no enlargement of either the liver or spleen.

On the evening of the day following admission, *i.e.* 6th October, the patient became suddenly collapsed, but with warmth and stimulant he improved. Next day, 7th October, he became worse again, and developed signs of meningitis. He was only semi-conscious, and it was doubtful if he could see. Both pupils were dilated, but reacted slightly to light, and there was a right internal strabismus. Some rigidity of the muscles of the back of the neck was also detected. Later on he became quite unconscious, irregularity of the pulse and respiration developed, and twitchings of the right side of the face were occasionally noticed. Lumbar puncture was performed, and the fluid withdrawn showed the presence of tubercle bacilli, and that the majority of the leucocytes were lymphocytes. The patient rapidly grew worse, and by the evening was quite comatose, and had to be nasally fed. The face was flushed and he perspired profusely. The pulse became imperceptible and the breathing was latterly of the Cheyne-Stokes type.

The blood was examined on two occasions, when a leucocytosis was detected—the leucocytes numbered on 7th October 19,700 and on the 8th October 13,400 per cent. No differential count was made.

The temperature, which registered 99° F. on admission, rose

slightly and gradually thereafter, and shortly before death registered 102° F.

Patient died at 5.30 P.M. on 8th October, three days after admission to hospital.

#### POST-MORTEM EXAMINATION.

The body is that of a much emaciated child. Rigor mortis is passing off.

*Brain.*—The membranes over the convexity are somewhat congested, while at the base there is a typical tubercular meningitis. This latter is in the form of a greenish yellow exudate, obscuring the structures between the crura cerebri and the optic tracts, and it extends to the superior surface of the cerebellum and into both Sylvian fissures, the opposing surfaces of which are adherent. Throughout this exudate numerous small sago-like bodies are observed along the course of the vessels. The cerebrum is markedly abnormal and presents rather a curious appearance. Some of the convolutions, in varying parts of their extent, are pale and swollen, and stand out distinctly above the others; they are densely hard to the touch, in marked contrast to the intervening soft and apparently normal convolutions. These sclerosed areas, which are very numerous—thirty such masses being present in the right hemisphere—are irregularly disposed, and vary in size between 7 sq. mm. and 25 sq. mm. They always retain the general contour of the convolutions, and their surface, from which the pia-arachnoid separates easily, is smooth, with occasionally a dimple in the centre. They are practically symmetrically distributed on the two sides, and are entirely limited to the cerebrum—the convexity, basal, and mesial aspects, none being detected in the pons, medulla oblongata, or cerebellum. On section the swelling and pallor of the abnormal convolutions are much more apparent; the fresh section, displaying a surface of a waxy appearance and almost entirely devoid of grey matter, contrasts very markedly with that of the normal convolutions (Fig. 1). This abnormal condition, as a rule, is most marked on the surface of the gyri, but in several places it implicates the grey matter in one or both sulci as well, and always extends for some distance into the white matter, so that an entire convolu-



tion may sometimes be so transferred. The ventricles are not dilated, nor are any sclerosed areas detected in their walls. No ependymal granulations are observed.

*Thorax.*—On opening the thorax a normal extent of the pericardium is exposed, and the pleuræ are found to be non-adherent.

*Heart.*—There is no evidence of pericarditis. The heart, which is slightly enlarged, is pale and flabby, and in the ventricular walls are numerous pale, hard masses. Two of these are much larger than the others, being about the size of walnuts, and situated in the wall of the right ventricle, while the remainder, which vary in size from that of a pea to that of a pin's head, are scattered throughout the substance of the left ventricular wall. Microscopic examination of these masses showed them to be composed of a peculiar form of vacuolar degeneration of the myocardium, which I have described elsewhere. There is no evidence of endocarditis, and both the pulmonary and aortic valves are quite competent.

*Lungs.*—Both lungs are the seat of a generalised tuberculous infection. In the left upper lobe is a large cavity with irregular ulcerated walls, and in the left lower lobe there is a tuberculous broncho-pneumonia. The mediastinal glands are enlarged and caseous.

*Abdomen.*—The mesenteric glands are enlarged and caseous. The liver is the seat of a tuberculous perihepatitis, and throughout its substance are numerous tuberculous foci. The spleen is also markedly tuberculous, and in both kidneys are miliary tubercles.

#### MICROSCOPIC EXAMINATION.

The tissues were fixed in a 3 per cent. solution of formaline for twenty-four hours, and thereafter were transferred to methylated spirit and embedded in paraffin. In addition to the ordinary staining methods, the nerve cells were studied in sections treated with Ehrlich's triacid stain and toluidin blue, the neuroglia by Ford Robertson's methyl violet method, and the medullated sheaths by Weigert's process.

Minute examination of the meningeal exudation verifies the naked-eye diagnosis of tubercular meningitis, tubercles with numerous tubercle bacilli being detected in the adventitia of the

vessels. In the enlarged and indurated portions of the convolutions, there is found a much denser felting of the neuroglial fibres than normal, and a slight numerical increase of the neuroglia cells, many of which are enlarged and contain two or more nuclei. The neuroglial fibres are coarser as well as more numerous than normal. This hypertrophy of the neuroglia is most marked, as a rule, on the surface of the convolutions, where sub-pial felting is present, and gradually diminishes towards the white matter and also towards the sulci, though here, too, it may be very distinct (Fig. 2). In these areas there is a great scarcity of nerve cells and medullated nerve sheaths, and at the same time it is observed that many of the nerve cells which remain are more deeply situated in the cerebrum than normally. This deficiency of nervous elements is most apparent about the centre of the convexity of the convolution, and the nerve cells become more numerous as the sulci are approached. Many of the nerve cells situated at the margin of the sclerosed area are very irregularly arranged with their apices pointing in all directions, instead of, as normally, towards the surface. Many are atrophied and they show all degrees of chromatolysis. Throughout these areas of gliosis and degeneration of nerve cells, there are, from the very surface of the convolutions to beyond the normal limits of the layer of nerve cells, numerous large pyramidal shaped and ganglionic-like cells; though scattered more or less throughout these areas, they, as a rule, tend to be arranged in groups. These cells are from two to four times the size of the largest pyramidal nerve cells normally found in the cortex, and many are similar in appearance to the multipolar cells met with in the anterior cornua of the spinal cord. In each of these abnormal nerve cells there is usually a large nucleus with bright nucleolus, and, in not a few, two or even three nuclei are detected (Fig. 3). The majority of these cells show few and small chromatic granules, which are usually arranged around the periphery, leaving a pale achromatic centre, though in some there is a normal amount of chromatic granules uniformly distributed and extending into the processes. The nuclei, which are frequently dislocated towards the periphery of the cell, occasionally show marked vacuolation, and the nuclei and nucleoli, as in normal nerve cells, are markedly oxyphilic in character.

In the sclerosed convolutions, which to the naked eye seemed quite normal, and in which there is neither any sub-pial felting nor any disturbance of the normal arrangement of the layers of nerve cells, groups of these previously described ganglionic cells are encountered deep down in the white matter. There is always associated with these accumulations of ganglionic cells a certain degree of neuroglial hypertrophy, similar in character to that met with in the sclerosed areas on the surface. Furthermore, these deeply situated areas, which contain no normal pyramidal nerve cells, but only the large ganglionic cells and a certain number of nerve fibres, are completely separated from the grey matter in the cortex by a band of normal white matter.

There is no sharp border to any of these abnormal areas, whether they may be situated on the surface of the convolutions or in the depth of the white matter. The abnormal gradually merges into the normal; the neuroglial processes get thinner and the network becomes less dense. In the case of the areas on the surface, on following the grey matter into the sulci, where it is, as a rule, less affected, concomitant with the diminution in the gliosis, more nerve cells are observed. At first they are very scanty and atrophic, and show all stages of chromatolysis, as also the irregular disposition previously referred to. Here and there a somewhat large but undoubted pyramidal nerve cell, with normal chromatic granules and containing two nuclei, is observed.

Throughout the sclerosed patches the blood-vessels are, if anything, less numerous than in the normal cerebral tissue. In the white matter at the periphery of the nodules widely dilated blood-vessels are occasionally present, while here and there are much dilated perivascular lymphatic spaces. At no part is there any evidence of round cell infiltration, and several sections of the sclerosed patches were examined for tubercle bacilli but with negative results.

#### COMMENTARY.

Clinically the case did not present any features of very special interest, but seemed merely an ordinary one of pulmonary tuberculosis terminating with meningitis. Neither the cardiac tumours nor the cerebral lesions were diagnosed during life, as there were no symptoms, other than those of meningitis, to

attract attention to grave abnormalities in either of these organs. At first sight it was thought, in view of the widespread tuberculous mischief, that the unusual cerebral lesions might be of a similar nature, but microscopic examination shows this not to be the case. In all these indurated and swollen portions of the convolutions there is a hypertrophy of the neuroglial tissue, as evidenced by the increased number and thickness of the fibrillar elements and the slight relative numerical increase of the cellular elements, some of which are much enlarged and contain two or more nuclei. There is also observed, concomitant with this sclerosis, a deficiency in number and also a degeneration of the nerve cells, and, further, there are invariably in these nodules numerous large pyramidal-shaped and ganglionic-like cells. That these large cells are nerve cells is shown by their shape, by the presence of processes, by the fact that the cells and processes contain chromatic elements, and by the presence of a nucleus with nucleolus, both of which are markedly oxyphilic in character. According to Levi (1) the nerve cell nucleus and nucleolus are chiefly oxyphilic in character, yet he describes in the nucleolus a few basophilic granules, which can be demonstrated in sections treated with Biondi's stain in weak solution. My sections were treated with Ehrlich's triacid stain, which, though showing the strongly oxyphilic property of the nucleus and nucleolus, fails to reveal the few basophilic granules described by Levi as being present in the nucleolus. Had these large cells only been of the pyramidal type, and had there been present some intermediate forms, one might have considered them hypertrophied or proliferating nerve cells. But their shape in some instances, their abnormal situation in others, and also the fact that no transitional forms between the cortical pyramidal nerve cell and the large ganglionic cell are visible, prevent us from accepting such an explanation. Moreover, many of these large cells show some degeneration in the form of chromatolytic change, at least an arrangement of chromatic granules not normal to the cortical nerve cell, though described by some authors as the usual disposition of the chromophile substance in the sympathetic ganglion (2) cells and in those of the column of Lockhart Clarke (3 and 4). This is also against the idea that these large cells are hypertrophied nerve cells, though Ford Robertson (5), while admitting that proliferation of nerve cells may take place, states that it

does not, however, lead to any stable regeneration. As the pyramidal nerve cells at the margins of these nodules are widely separated, irregularly placed, and in various degrees of degeneration, and also as the transition from the normal to the abnormal is more or less gradual, one may conclude that this gliosis is gradually extending and infiltrating the surrounding healthy cerebral tissue.

This combination of gliosis and ganglionic cells makes up the picture of ganglionic neuroglioma, a very rare form of tumour. Most authors consider this tumour to be of congenital origin, and such a view is the only way in which we can satisfactorily account for the presence of ganglionic nerve cells in regions where ganglionic nerve cells do not normally exist.

Ziegler (6) describes the condition of "Ganglionic Neuroglioma" as "an apparent enlargement of some portion of the brain not marked off by any definite boundary from the surrounding tissue, or as a more circumscribed nodose tumour. The pia mater overlying the enlarged portion is not altered, and the configuration of the gyri is, in general, left intact. On transverse section the difference in tint, normally so striking, between the cortex and the medullary white matter is indistinct or entirely absent: the tissue looks uniformly white or greyish white. It is of firmer consistence than the normal tissue, and sometimes is firm and tough in texture. The matrix of the growth consists of neuroglia similar in character to that of the patches in disseminated sclerosis; it is sometimes dense and firm, sometimes loose in texture. The tissue contains ganglion cells, not only in the region of the original cortex, but also within the white matter of the gyri and the centrum ovale; these cells are loosely scattered or aggregated in groups. Medullated nerve fibres are visible only in some parts of the tumour, but they never approach in size or number the fibres that are normally contained in the white matter of the brain." It will be noticed how closely the cerebral lesions in my case resemble this description by Ziegler.

Stengel (7) states that these tumours as a rule are multiple, and are present as numerous "nodular condensations throughout the brain." This fact of the multiplicity of the growth is not mentioned by any other author, so far as I can find.

This condition is most likely to be mistaken for gliosis or a glioma, more especially perhaps when infiltrating an area where

ganglionic cells normally exist, as, for example, the basal nuclei of the brain. Indeed, Thoma (8) states that there is no such individual or characteristic tumour as a "ganglionic neuroglioma," and that such appearances are always produced by the infiltration of a cerebral nucleus either by sclerosis or a glioma. This may be true in some cases, but assuredly it is not so in such as mine, where ganglionic cells are found in regions where they do not normally exist. It must not be forgotten, however, that in the motor regions of the human cerebral cortex are nerve cells so large and so irregular in shape that Bevan Lewis (9) has proposed to call them "ganglion cells," and the layer which they form "the ganglion cell layer of the cortex." This author considers them the cells which have to do with motion, and, as they are specially numerous and large in the regions which govern the movements of the lower limbs, that they are proportionate in size to the amount of muscular energy requiring to be expended. Betz (10) called them the "giant cells," and at one time they were supposed to be pathological. Though found all over the cortex, they are most numerous and largest in the motor regions. They are situated immediately below the layer of large pyramidal cells, and superficial to the fusiform layer of cells. "They are irregularly distributed or in clumps; they vary in size and shape, and have a large oval nucleus" [Bevan Lewis (11)]. "Some of them resemble the motor nerve cells of the spinal cord" [Gowers (12)]. "These cells are most typically ganglionic in shape in man, but in the lower animals are definitely pyramidal in shape" (Bevan Lewis). However, all the figures and diagrams, which Bevan Lewis and Gowers give, show these cells as fairly definitely pyramidal in shape, and comparable to the other elements of the cortex. In my case, on the other hand, there is no gradation between the pyramidal nerve cells and the large ganglionic cells, the latter being frankly ganglionic in shape; and further, they do not bear any constant relationship to the layer of nerve cells, and are entirely limited to the areas of sclerosis, whether situated on the surface of the convolutions or deep down in the white matter, and whether the sclerosis occur in the motor region of the cortex or not.

It is generally recognised that it is very difficult to differentiate between gliosis and a glioma, as the two lesions may have exactly similar appearances, both macroscopically and microscopically.

Gliomata may be very cellular and exceedingly vascular, or poor in cells and vessels, and all gradations are met with between these two varieties. Gowers (13) states that the fibrous form of glioma has often been mistaken for sclerosis. Ziegler (14), while mentioning the difficulty of diagnosing between gliosis and glioma, at the same time remarks that it is equally difficult to differentiate between gliosis and ganglionic neuroglioma. To my mind this cannot be so, at least in the majority of cases, if it be true, as he himself implies, that the tumour is most usually found on the surface of the brain. In my opinion the presence of ganglionic nerve cells in abnormal situations removes the pathological condition entirely from the sphere of sclerosis. Ganglion cells, excepting of course these large irregular pyramidal cells described as "ganglion cells" by Bevan Lewis, are not normally found in the grey matter of the cortex nor in the white matter of the cerebrum, so that their presence in these situations, as that of cartilage or bone cells, relegates ganglionic neurogliomata, equally with chondromata and osteomata, among the new formations; and, moreover, among these new formations called congenital inclusions.

Nerve cells proliferate only in very rare instances, in fact until recently proliferation of nerve cells was greatly disputed, and it was only in 1896 that Levi (15) conclusively proved that such a phenomenon did occur, though only to a slight extent. Further, Stengel (16) and Coats (17) state that ganglionic neuroglioma has been found in the suprarenal body, and if this be so, it lends additional support to the view that ganglionic neuroglioma is a new formation, and not a gliosis or glioma modifying the cortex or some other region of the brain. When one recollects that the suprarenals in part are developed from the sympathetic ganglia, it is quite intelligible how a tumour composed of nerve fibres, ganglion cells and neuroglia, in short, a ganglionic neuroglioma, can arise in such a situation. Consequently, it is most likely that these tumour areas arise by a dislocation of nerve cells, probably from groups of those of the ganglionic type. There is the possibility, however, that they may be derived from the cortex, though in this case their characters must have been much modified owing to the altered surroundings.

Though there should not, as a rule, be much difficulty in

differentiating between ganglionic neuroglioma and an ordinary glioma or gliosis, there is some risk, at least from a naked-eye examination alone, of confounding the first-mentioned condition with what has been termed "tuberous or hypertrophic sclerosis of the cerebral convolutions" (18). The microscopic appearances of the two conditions are very similar, tuberous sclerosis being also characterised by numerous pale hard swellings of portions of the convolutions scattered irregularly over the cerebrum. In its minute structure, however, it differs materially from ganglionic neuroglioma in an absence of ganglion cells, which, as I have said before, are typical of that form of tumour. Microscopically tuberous sclerosis shows a complete absence of nerve cells and medullated sheaths, the tissue being composed of a dense avascular neuroglial feltwork.

In spite of the radical difference between these two conditions, which, moreover, are both congenital—tuberous sclerosis being due to an absence of normal tissue, and the other to the presence of abnormal tissue—Thibal incorporates in his thesis, "*Sclérose Tubéreuse*," a case diagnosed and reported as such by Brückner (19), but which, to my mind, seems an example of ganglionic neuroglioma. Although Thibal records it in full, he doubts if it really be a case of hypertrophic or tuberous sclerosis, as it differed from that condition not only in the clinical history, but also in the microscopic characters of the lesion; consequently, it may not be out of place here to give a short summary of Thibal's translation of Brückner's report.

The patient was a female who died at the age of twenty-two years. From infancy she had been mentally deficient, and when nine years of age commenced to suffer from epileptiform convulsions. Later on she became maniacal and was removed to an asylum, where she developed pulmonary tuberculosis and died within a few months. At the autopsy there was found extensive tuberculous disease of both lungs and of the intestinal tract. The brain was enlarged and pale, and over the surface of both hemispheres were numerous large hard prominences representing hypertrophied parts of the convolutions. These abnormal areas had a rounded shape with a smooth surface, in the centre of the largest of which was usually a dimple, and they were sharply demarcated from the surrounding healthy convolutions. The pia mater, which showed neither thickening nor loss of trans-



parency, was easily separated from both the normal and abnormal convolutions. In the cerebellum there were two nodules of a similar nature of about the size of a hazel-nut. Both lateral ventricles were dilated. On the ventricular aspect of the corpora striata and optic thalami were numerous small rounded nodules of a white colour, and varying in size from that of a pin's head to that of a small pea. Those situated towards the foramen of Monro were polypoid, with a soft consistency and granular surface. On section of the sclerosed areas in the cerebral convolutions, it was observed that the transformation affected almost solely the grey matter of the cortex. These areas had a hard consistency and were opaque. The contrast between the grey and the white matters was very indistinct, especially at the summits of the convolutions, where the condition was most marked. In the largest nodules it was observed that the layer of grey matter was thicker than normal, being 5-7 mm. in thickness, while at the umbilicated parts it only measured 2 mm.

On microscopic examination of the sclerosed areas it was found that there was a great neuroglial hypertrophy, more especially of the fibrillar elements, and on the surface of the altered convolutions there was always marked sub-pial felting. On passing into the white matter the neuroglial felting was less dense, and round cells and occasionally angular cells were observed. Here and there was seen a large irregular multipolar ganglionic cell, with a large nucleus which was swollen, and in some cases, vesicular. Occasionally these large cells were grouped together. The pigmentation of these cells and their processes was little modified. The blood-vessels in these areas were engorged with blood, and possessed in many instances widely dilated perivascular lymphatic spaces. Round the cortical vessels there were congregated round cells, which did not seem in relationship, by means of processes, with the vessels. The ependymal polypoid masses were composed of a fundamental fibrous stroma, with numerous spaces filled with large round cells. Towards the margin were some calcareous bodies similar to what are found in the normal pineal body—in fact, the general structure of these nodules simulated somewhat closely that of the above-mentioned gland. These ependymal granulations Brückner considered inflammatory in origin and quite distinct

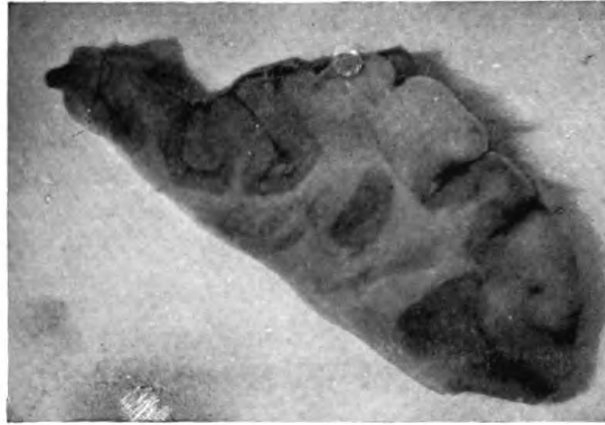


FIG. 1.



FIG. 2.

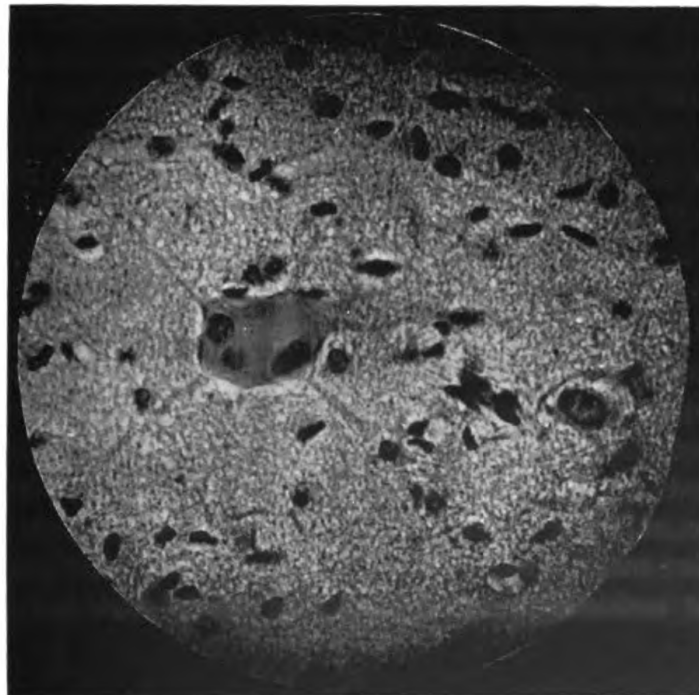


FIG. 3.



from the cortical change. The latter he considered of the nature of a hypertrophic sclerosis, and while comparing his case with this lesion discusses the question of vascular supply and gliosis, but in his argument omits to take into consideration the presence of the ganglionic cells. These are not present in sclerosis, and, as I have previously remarked, show the true nature of the lesion.

Bourneville and Brissaud (20), whose opinion Thibal expresses in his thesis, think, from the presence of round cells and the engorgement of the vessels, that Brückner's case might be an earlier and inflammatory stage of the sclerotic change, but make no mention of the ganglionic cells described by Brückner in his microscopic examination. It must not be forgotten, however, that probably many of the round cells Brückner describes were really gliomatous in nature, since it is only within recent years that it has been possible, by means of modified staining processes, to demonstrate the intimate connection between neuroglial cells and their ramifications, and so to discriminate between round cells and glial cells.

The only differences between Brückner's case and my own are the presence of tumours in the cerebellum and the degree of vascularity of the abnormal areas. The vascularity of a tumour we know is a very variable quantity, and perhaps the avascular condition of the tumours in my case accounts for the chromatolysis of the ganglion cells, a condition which was absent in the case described by Brückner. Thus I do not only entertain Thibal's (21) doubts, but go further, and express the opinion that Brückner's case was really an example of ganglionic neuroglioma.

Ziegler, in his bibliography of ganglionic neuroglioma, mentions a case reported by Otto (22) in *Virchow's Archives*, and this author, in his paper, refers to four other cases of a similar nature put on record by Simon (23). All these cases were in adults, and in no instance did the presence of the tumours give rise to any nervous symptoms during life, and the description of their minute anatomy shows that they do not conform in any point whatsoever to the condition of "ganglionic neuroglioma."

It seems, therefore, apparent from the above that there is such a distinct and individual tumour as ganglionic neuroglioma, and that it has the following characteristics. In origin it is congenital and, though most frequently situated in the brain, may

occur in the suprarenals. In contradistinction to ordinary glioma the process usually commences on the surface of the cerebral convolutions, without, however, involving the meninges, and persists as numerous hard and pale swellings of the same. As a rule the contour of the convolutions is not disturbed, and the surface is smooth. Histologically, a ganglionic neuroglioma presents a variable degree of vascularity and is composed of a hypertrophied neuroglial matrix, throughout which are scattered, singly or in groups, large ganglionic nerve cells.

In conclusion, I have to thank Dr Geo. S. Middleton for allowing me to report this case. My thanks are also due to Prof. R. Muir of Glasgow University for granting me permission to work in his laboratory, and for much valuable advice. To Drs Cowan and Ferguson I am also indebted for hints regarding pathological technique and the interpretation of microscopic appearances.

#### DESCRIPTION OF PLATE.

- FIG. 1.—Photograph of a section through the occipital lobe of brain, showing the swelling and absence of grey matter in the abnormal convolutions.
- FIG. 2.—Section of brain passing through a normal and an abnormal convolution, showing the neuroglial hypertrophy in the form of sub-pial felting in the sclerosed convolution. There is a sulcus with normal pia mater between the two convolutions. (Stained by Robertson's methyl violet method.)
- FIG. 3.—Photo-micrograph of one of the large multipolar ganglionic cells with three nuclei occurring in a sclerosed convolution.

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### PROGRESSIVE MUSCULAR ATROPHY.

By LEONARD WILLIAMS, M.D., M.R.C.P.,  
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IN connection with those forms of muscular atrophy to which the adjective "progressive" has been applied, two problems seemed at one time to present themselves. The one was whether the pseudo-hypertrophic form was essentially different from the purely atrophic forms; and the other, whether the spinal cord, being the undoubted seat of the mischief in some, could rightly be held responsible in all. The difficulties associated with the first of these problems seemed gradually to disappear as the second approached solution. The differentiation between progressive muscular atrophy which was demonstrably due to changes in the anterior horns of the cord on the one hand, and, on the other, the forms of atrophy in which no such changes could be discovered, led not only to the establishment of definite clinical distinctions between the two, but it aided in the realisation of the fact that there was no essential difference between the pseudo-hypertrophic and other types of non-spinal atrophy.

The experience of most observers has had the effect of deepening the general conviction of the essential relationship between the various forms of myopathy or muscular dystrophy, the occurrence of a pseudo-hypertrophy being regarded merely as

an incident which may or may not arise in the development of any of the recognised types. Curiously enough, however, the broad line which for some time separated spinal atrophy from myopathy has shown several signs of narrowing, and the trend of opinion seems to be in the direction of holding the spinal cord responsible not for progressive muscular atrophy only, but also for myopathy. It is not necessary to go so far as Osler ("Pract. of Medicine," 4th Ed., p. 936), who says that "the whole question is in a chaotic state," to realise that the distinctions hitherto relied upon are not always trustworthy, or that there may conceivably be cases in which the two forms are present at the same time.

At a clinical evening of the Neurological Society on February 26, 1903, which was devoted exclusively to cases of myopathy, several instances of each of the recognised types were exhibited, together with some anomalous forms (*Brain*, Vol. xxvi. No. 101, Spring 1903, p. 141). In the discussion which followed, which is unfortunately not reported, a very close ætiological relationship between all the forms of muscular dystrophy was generally admitted, and a large number of the speakers seemed to incline very strongly to the view that although the pathology of the condition is at present very obscure, it will eventually prove to be associated with some lesions of the cord which are not discoverable by the means of investigation at present open to us.

Against this view it was pointed out, by Dr Beever, if I remember rightly, that the grouping of the affected muscles in the primary myopathies does not correspond to that which is found in undoubted disease of the cord, and that the diagnostic points between typical cases of myopathy on the one hand and progressive muscular atrophy of spinal origin on the other, seemed to be multiplying rather than diminishing. While there is no gainsaying either the truth of these observations or the great weight which they must always exercise in any estimate of the possible responsibility of the spinal cord for what is now conveniently termed myopathic degeneration, yet it is undoubtedly true that cases sometimes arise in which absolute reliance upon text-book distinctions between the myopathies and progressive muscular atrophy would, to say the least, give rise to confusion. These distinctions are commonly given as: (1) The age of the patient and the family history; (2) The presence or absence of fibrillary twitchings; (3) The distribution of the affected muscles,

and the order of their involvement. There are others, of course, which vary in number with the size of the text-book and the amount of space which is devoted to the subject, but it is upon these three that the chief stress is usually laid.

Some of the cases exhibited at the above-mentioned meeting, notably one by Dr Risien Russell and another by Dr James Taylor, show that the first two of these points are by no means always to be relied upon, and I desire to record the particulars of a patient whom I demonstrated at the Polyclinic in March 1905, which suggest that even when the diagnostic features presented under these three heads combine to point in one direction, their consideration to the exclusion of others might easily give rise to an erroneous conclusion.

A negative family history is, of course, valueless, but the age of onset of the symptoms is regarded as an important point in the differential diagnosis. Progressive muscular atrophy is a disease of adult life which is seldom or never seen in the very young; whereas the myopathies are essentially congenital, showing themselves as a rule in childhood, and seldom delaying their appearance beyond the age of puberty. Dr Risien Russell's case was an exception to this rule. The patient was a man of 46, a coal porter, who had first noticed weakness in his right arm after sunstroke, three and a half years before. This would make his age at the onset of the symptoms 43 years—a very exceptionally late beginning for a case, which was otherwise a typical muscular dystrophy, with nothing to suggest spinal involvement. There was no similar affection in other members of the same family.

The presence of fibrillary twitchings is so common in progressive muscular atrophy, and so uncommon in any form of myopathy, that it is always mentioned as a diagnostic point of the utmost value. Dr James Taylor's case (*ibid.* p. 151) was that of a child, aged 11, who had "very distinct general twitchings in the muscles all over the body, especially well marked in the pectorals"; and so unusual did he regard this phenomenon in association with what was undoubtedly a myopathy, that Dr Taylor adds to his description the remark, that "the muscular twitching suggests the possible association of spinal cord disease with the myopathy."

In connection with the distribution of the affected muscles and the order of their involvement, it is usually laid down that



it is the proximal segments of the limbs which first show the wasting in myopathy, and that the distal segments escape until a very late period; whereas in the spinal affection it is the small muscles of the hand and forearm which first show signs of weakness and atrophic change. Early involvement of the latissimus dorsi, of the trapezius, and of the pectoralis major in its lower part, is regarded as almost pathognomonic of a muscular dystrophy; whereas preservation of these muscles, more especially of the trapezius (in its upper part, at any rate), is strikingly suggestive of the condition being due to spinal involvement.

The case which I have to record is that of a man, 28 years of age, in whom no family tendency could be traced. The muscular wasting dated from 1903, a year after he had fallen off a shed about 8 feet in height. The muscles to be first affected were those of his left arm, both upper arm and forearm being involved, and, to some extent, the interossei of the hand on the same side. He next noticed that his chest muscles twitched and began to waste. The small muscles of the feet then became affected, and later, the thigh muscles on both sides. On examination, he was seen to have a very definite myopathic facies, though, on testing, there seemed to be no weakness of any of the facial muscles, except, perhaps, the orbicularis oris. The orbicularis palpebrarum was quite intact. Fibrillary twitchings were present in most of the muscles above the waist, and were particularly noticeable in the pectorals and in what remained of the triceps on both sides. There was very decided wasting of the lower part of the pectoralis major, of the latissimus dorsi, of the biceps and the triceps. The trapezius, however, and the serratus magnus had altogether escaped. None of the muscles were conspicuously hypertrophied, but the calf muscles on both sides appeared harder and firmer than normal. The knee-jerks could not be obtained, a fact which did not seem to be sufficiently accounted for by the degree of involvement of the quadriceps extensors. There was no sphincter trouble, and no R.D. to electrical testing.

The importance of this case resides in the fact that although it was undoubtedly one of muscular dystrophy, it failed to conform to the text-book picture of this condition in the three diagnostic points already alluded to. The age did not present so striking a departure from the majority of cases as did Dr

Risien Russell's patient, but a muscular atrophy of a progressive nature beginning at 28 years, regarded as an isolated fact, suggests not a myopathy, but an atrophy of spinal origin. The symptoms in Dr Russell's case followed on "sunstroke," and those in the above, on a fall off a shed, so that it seems probable that in both, trauma may have had some influence in provoking a degenerative process which might otherwise have lain dormant.

The fibrillary twitchings in my patient varied very much both in distribution and in intensity from time to time. When I first saw him they were very marked, especially in the pectoral muscles, but when he was exhibited at the Polyclinic they were feeble and confined to the triceps. They were, however, very easily elicited.

The distribution of the affected muscles and the order of their involvement in the above case represents a sufficiently noteworthy departure from what is generally considered typical. For the order one has of course to depend upon a patient's statements, but my patient was intelligent and observant in a degree beyond what is usual in the hospital class (he had travelled much and had seen men and cities), and he was quite positive that the small muscles of his left hand were very early involved, some time in fact before the twitchings in the chest muscles, which heralded their wasting, became at all noticeable. The persistence of the trapezius in the presence of so much wasting in the immediate neighbourhood was especially remarkable. This muscle is not only one of the first to waste in typical cases of myopathy, but it is of all others the one which as a rule escapes to the very end in muscular atrophy of spinal origin.

## **NEW YORK STATE AND PSYCHIATRIC TEACHING.**

C. MACFIE CAMPBELL, M.B., Ch.B.

IN Great Britain the question of the treatment of the insane is arousing considerable interest at the present moment. It is being forced on the professional mind that it is not sufficient to clothe, house, feed, or even entertain the insane, but that medical responsibility goes still deeper: the brain disease, which

manifests itself by mental symptoms, requires the same careful study by specially trained physicians which is given to the other special branches of medicine.

The ophthalmologist, the surgeon, the neurologist, are expected to have an expert knowledge of the diseases which they deal with, and to use the material which they have in the solution of the questions of the origin, nature, and treatment of disease. The alienist is in addition expected to be an expert business manager, and to find time in the intervals of a busy administrative life for his proper medical work ; to denote this additional requirement the physician in charge of a hospital for the insane is called a medical superintendent.

Even before he finds his medical work hampered by his administrative duties, the student of mental diseases has reason to envy workers in other fields. While they have no difficulty in obtaining a special training in clinics organised to meet their needs, he finds in Great Britain no psychiatric clinic where he may learn from a recognised teacher the special methods required in his work, and receive guidance as to the issues to be met and the most profitable lines of investigation.

Whatever handicaps the physician means a loss to the patient ; the patient here is one whom we are bound to treat with especial consideration, for he is no longer able to look after his own interests. The State deprives him of his liberty, and compels him to undergo treatment by a physician whom he himself has no voice in choosing. To give the physician no opportunities for the special study of his particular subject, and to encumber him with non-medical administrative duties, is a handicap to the physician and a serious injustice to the insane.

The responsibility for this injustice is being felt by some of our medical schools, and in a short time it is hoped that the opportunities for the study of mental diseases will be satisfactory. In Germany the universities have followed the example set by Heidelberg thirty years ago, and the university psychiatric clinics have done much to give German psychiatry its present pre-eminence. In Great Britain there are hopeful signs in the special observation wards for the insane in the Eastern District Hospital, Glasgow ; in the Edinburgh plans for a psychiatric clinic in the Royal Infirmary ; and in the reception wards for the insane which certain London hospitals propose to institute.

In America, the University of Michigan has taken steps to open a psychiatric clinic. In New York State the initiative has been left by the universities to the State, and the enlightened policy of the State in this respect should quicken our own feeling of responsibility.

I do not propose to consider the organisation of the hospitals for the insane, of which the State is justly proud, but merely to call attention to the central Pathological Institute, and more especially to the clinical department, which is essentially a psychiatric clinic, a centre of clinical instruction and scientific research.

New York State has to care for between 26,000 and 27,000 insane inmates of its 39 public and private asylums; leaving aside the private asylums there are 14 State hospitals, whose staffs amount to 155 physicians. The entire supervision of all these institutions lies in the hands of the Lunacy Commission, consisting of one medical man, one lawyer, and one layman. The Lunacy Commission, in its Sixth Annual Report, recommended the establishment and maintenance of a pathological laboratory or institute which should be a department of the State hospital system. The Pathological Institute was accordingly founded in 1896; at first its function was similar to that of the Pathological Laboratory of the London County Asylums, and of the Laboratory of the Scottish Asylums. In 1900 a committee of superintendents, in a report on the relation of a central institute to the hospitals, stated: "We believe that the Central Institute could be of great advantage to the hospitals by offering to the men in the State service instruction in advanced clinical methods such as could be obtained in a psychiatric clinic." For this purpose a special hospital service was attached to the Institute, which now came into closer contact with the clinical work in the various hospitals. Since the organisation of the psychiatric clinic or clinical department of the Institute, over a hundred assistant physicians have been enabled to follow a course of clinical instruction. As a rule the course is of three months' duration, and embraces, besides clinical work, lectures on the anatomy of the nervous system, post-mortem work, and discussions of current literature and special topics. Professor Adolf Meyer, Director of the Institute, outlined the scope of the clinical work: "to show by example the methods and issues of

case-taking, examination (mental and physical), and continued observation of cases, and the final working up and grouping. . . . To show, on special material, the chief data of neurology and psychiatry needed for work on the insane, such as is not usually taught in medical schools; and to point to a profitable utilisation of the records which are accumulated."

The Institute occupies a building on Ward's Island, New York, and is in close connection with one of the large State hospitals situated on the island. The clinical material is plentiful and varied, and is ample for the seven or eight physicians attending the clinic at any one time. Patients are allotted on admission, and a thorough mental and physical status is taken: special attention is paid to the nervous and cardio-vascular systems. The physicians' notes are immediately typewritten, and a neatly typewritten record is a pleasant contrast to the usual manuscript. In most of the State hospitals typewriters are available, and the physicians are thus saved a large amount of time. Each patient is presented by the physician who has taken the case before the clinic; the case is discussed by all, extra points to be ascertained are suggested, and the lines of further investigation are determined. For presentation of the case, each physician prepares a summary, which is a critical digest of the case, and in which is embodied all points used in the diagnosis of the psychosis. Professor Meyer lays great stress on the preparation of such a summary as soon as the case has been thoroughly examined, and even although the clinical picture varies rapidly, for in the preparation of such a critical digest the omission of necessary data is at once seen and can be rectified before it is too late.

At the end of each course the cases are grouped in a provisional way, and each physician examines one of these groups and presents to the others the conclusions which he draws from his material.

The teaching is by no means dogmatic, hasty classification is discouraged, and the understanding of the pathogenesis of the psychosis is looked upon as essential, while the verbal diagnosis is subsidiary. The classification of Kraepelin, whose teaching has been received with great enthusiasm in America, and whose views are better known than those of any other foreign author, is used as furnishing a useful basis.

One point that is of great importance is the care that is devoted to getting an accurate anamnesis; when it is considered advisable, and this is frequently the case, a visit is paid to the home or work-place of the patient, and this first-hand information on the environment of the patient is of great value.

By the foundation of the Pathological Institute with its psychiatric clinic, New York State has given a powerful impulse to the study of mental diseases, and shown in what a wide sense it understands its responsibility towards the insane; the teaching, however, which it offers, is only open to those already in the State service.

The exact method which it has taken to accomplish its end has been largely determined by the organisation of the State hospital system. In Great Britain circumstances are very different, but it is hoped that the same progressive spirit which has caused New York State to offer special psychiatric training to its medical officers, will lead in Great Britain to the foundation of university psychiatric clinics analogous to other special clinics.

## Abstracts

### ANATOMY.

**THE GENESIS OF THE PROTOPLASMIC PROCESSES OF THE**  
 (179) **NERVE CELL.** (Su la genesi del prolungamenti protoplas-  
 matici della cellula nervosa.) FRAGNITO, *Annali di neurologia*,  
 Anno xxii, Fasc. 4.

IN the cells of the cerebral cortex of the embryos of mammals the author, in 1899, noticed nuclear structures in the dendrites which suggested an origin of the protoplasmic processes analogous to that of the axis-cylinder. This interesting point has been developed in this later investigation.

In the first place, contrary to the opinion expressed by Ramon y Cajal that the protoplasmic processes appear in the embryo of the chick on the 3rd, 4th, or 5th day of incubation, and by Bethe that they appear about the 67th hour, the author has found that they cannot be recognised before the 7th day, and he has seen them still in the process of formation up to the 18th day. Bombicci also says that he has not seen them before the 6th day.

In 1902, Fragnito criticised the views of Cajal, and said that the processes described by him had no relation of continuity with the neuroblasts, and that they really belonged to the "myelospangium" of His. Between the processes described by His, Cajal, and Bethe, and those spoken of by the author, there is not only a difference of size and of structure, but also in their mode of formation.

In the processes of a cell (figured in this article) taken from the anterior cornu of the spinal cord of a chick on the 10th day of incubation, nuclei could be seen lying within the processes. In one there were five nuclei whose long axis lay in the long axis of the process. Near the cell these were more round and closer to each other. That they were not simply lying against the cell process was proved by the facts that they were lying in the same plane as the intermediate protoplasm, that the processes were broader at the level of the nuclei, and that the nuclei were enveloped by neurofibrils.

The analogy between the formation of these processes and that of the axis-cylinders is complete, and eliminates every histogenetic difference between them. The same chain of cells precedes the

development of both, and analogous changes of these cells can be followed in all their phases.

But there is no analogy between these processes and those filaments which are met with in the first stages of development, and they can be easily distinguished from these latter in all stages. Moreover, it has been established by Paladino, and by Capobianco and Fragnito, that the neuroglia fibres envelop and also penetrate into the nerve cells, and it is probable that this relation is established in the early stages of development.

The true dendrites are always less numerous than the false dendrites, and it has been known that the dendrites diminish in number as the cell approaches its full development.

Ramon y Cajal has attempted to explain this fact by suggesting that in the early stages of development the cell sends out processes in all directions, but only those which succeed in establishing a relation of contiguity with the axis-cylinder of another cell survive; the others atrophy and disappear. The author holds that this has never been proved, and that the apparent diminution depends on the distinction being made between the true and false dendrites, when the cell is sufficiently developed to be clearly outlined from the surrounding structures. R. G. ROWS.

#### ON THE ARCUATE NUCLEI OF THE MEDULLA OBLONGATA

(180) AND ON ABERRANT ACCESSORY OLIVES. (Ueber die

Nuclei arciformes der Medulla oblongata und ueber accessorische Nebenoliven in derselben.) G. VOLPI-GHIRANDINI, *Neurol.*

*Centralb.*, March 1, 1905, p. 196.

THIS paper adds little to our knowledge of the anatomy of this region. It contains the report of a case in which an exceptionally large nucleus arciformes was found; this extends along the periphery of the cross-section of the medulla from the ventral surface of the pyramidal tracts to the neighbourhood of the spinal trigeminal root, being at some levels divided into two or three segments by the external arcuate fibres. In this case a strand of pyramidal fibres was separated off by it. There were also collections of cells ventro-lateral to the hypoglossal nuclei, which resembled the inferior olives in structure. These are regarded as aberrant accessory olives.

The very variable size of the nuclei arciformes may be explained by the fact that they are structures peculiar to man, and consequently not definitely fixed. They occasionally extend without interruption into the pontine nuclei, which they resemble in structure and perhaps too in physiological significance.

GORDON HOLMES.



**ON THE ARCUATE FIBRES OF THE MEDULLA OBLONGATA.**

(181) (*Ueber Fibræ arciformes medullae spinalis.*) L. JACOBSON,  
*Neurol. Centralb.*, April 1, 1905, p. 295.

THE various categories of *fibræ arcuatæ* of the medulla oblongata have homologues in the human cord, especially in its sacral region.

1. *Fibræ arciformes superficiales ventrales*—a band of fibres which runs in the ventral periphery of the cross-section of the sacral cord. The fibres can be traced, though generally interrupted in their course, from the anterior commissure. They gradually end in their course by turning to run longitudinally in the ventral columns.

2. *F. arciformes superficiales laterales*.—These run from the lateral bundle of the dorsal roots across Lissauer's zone and as far ventralwards as the middle of the lateral columns. These bundles too branch up in their course. They seem to be dorsal root fibres, but there is no definite proof of them being so, though no other origin can be determined for them.

3. *F. arciformes superficiales dorsales* also exist chiefly in the middle sacral cord. They run along the dorsal medium fissure—these belong to the dorsal columns—and in the dorsal periphery of the dorsal columns. The latter are dorsal root fibres, which run chiefly to the septum paramedianum, where they enter the dorsal columns.

4. *F. arciformes profundæ latero-ventrales*.—These were best observed in the chimpanzee; they are present, but not so compact, in man. They arise in the lateral ground layer of the sacral cord, and run as a fairly compact bundle round the lateral surface of the ventral horn to disappear in the ventral root zone. They probably spring from the intermediate grey matter of the cord.

A critical review of the literature of the subject concludes the paper.

GORDON HOLMES.

**PSYCHOLOGY.****A BIOLOGICAL THEORY OF SLEEP.** (*Esquisse d'une théorie*

(182) *biologique du sommeil.*) ED. CLAPARÈDE, *Arch. de Psychol.*,  
 Feb.-March 1905, p. 246.

SLEEP may be studied from a biological as well as from a physiological point of view. To the biological aspect of the subject, M. Claparède devotes 100 pages of the *Archiv. de Psychologie*. After discussing and rejecting the classical theories of the significance of sleep, the writer comes to the important conclusion that sleep is an active, positive function, and not a consequence of exhaustion

Seeing that sleep, in normal circumstances, precedes exhaustion, and that exhaustion frequently causes insomnia, the inference is drawn that sleep is a defensive function, an instinct which, by depriving the animal of energy, prevents the occurrence of the state of exhaustion.

By this conception we can easily explain all those facts which are quite inexplicable by the usual passive (toxic, chemical) theories; such facts, namely, as the want of parallelism between sleep and exhaustion, the periodicity of sleep, the possibility of retarding sleep by interest or by will, the production of sleep by suggestion, partial sleep, the variety of types of sleep in animals. Hibernation also we may regard as a kind of sleep derived from sleep of the ordinary kind, and of which the type is probably due to secondary adaptation.

As to the mechanism of sleep, it can be shown that sleep only comes on and persists and becomes complete in proportion as the interest of the subject in external circumstances or in his own thoughts becomes less intense than the instinct of sleep itself—sleep is a reaction of loss of interest in the present situation. It constitutes a sort of psychological suicide. When one goes to sleep, one renounces *ipso facto* one's perception of the external world, one's activity, one's adaptability, in a sense, indeed, one's life.

The reparatory effect of sleep arises, firstly, from the rest, the organism profiting by the arrest of muscular activity to get rid of waste products; and, secondly, by an increase in the trophic or assimilative processes, the relaxation of the "mental tension" being probably compensated for by an increase in the "vegetative tension."

In conclusion, the author argues that this conception of sleep may lead to a biological conception of hysteria, the two conditions being analogous although not identical.

W. B. DRUMMOND.

## **PATHOLOGY.**

**A PATHOLOGICAL STUDY OF ACUTE MYELITIS.** J. W.  
(183) RHEIN, *Univ. of Penn. Med. Bull.*, Jan. 1905, p. 373.

Two cases are recorded. A married woman (in whom syphilis was excluded) began to suffer from burning pains in the epigastrium and vomiting. Pain in the legs, tingling and formication up to the umbilicus, weakness of the legs going on in a few hours to complete paralysis then came on, and were accompanied by loss of sphincter control. On the tenth day there was extension to the hands of the tingling, the face and head becoming involved later. She died on the twentieth day.

The main changes found post-mortem were round-celled infiltration of the pia mater throughout, foci of inflammation in the cord, thickening of some of the blood-vessels in the foci, surrounded by round-celled infiltration and occasionally hæmorrhages, and destruction of the nerve elements in the foci. The Marchi method showed recent and intense myelin degeneration.

The second case was a single woman 39 years of age, with no history of syphilis. She rapidly developed a total anæsthesia below the sixth rib, with paraplegia and incontinence. On the ninth day the paralysis spread to the arms, and in three days the arm paralysis became total. She died four days later.

The Müller hardened cord was stained by the Weigert and Marchi methods, and some sections were stained by carmine and fuchsin. The anterior horn cells of the thoracic and lumbar regions were swollen, but those of the cervical enlargement were normal. No degeneration could be found by the Marchi stain in the myelin sheaths. There were no evidences of round-celled infiltration anywhere.

R. discusses these cases, and gives a *précis* of the views of most of those who have reported similar cases. He classifies them into the two categories, the first being the more numerous, and being inflammatory, showing involvement of blood-vessels and cellular infiltration: whilst the second show none of the pathological signs of inflammation, this class being much the smaller.

A good bibliography is given.

STANLEY BARNES.

**THE PROCESS OF REGENERATION IN AN AFFERENT  
(184) NERVE.** HENRY HEAD and C. E. HAM, *Journ. of Physiol.*,  
Feb. 1905.

IN a preliminary communication, the results of the division and complete isolation of the radial nerve of an adult cat are described. Provided that strict precautions are taken against union with other trunks, the radial nerve 540 days after division is found to be in a resting condition, and consists of spindle-shaped fibres with elongated nuclei. If united to another nerve when in this resting condition it is at last completely restored. The isolated nerve was successfully joined to the central end of the cut median. Shortly after union the spindle-shaped cells lengthen and form definite fibres without an axis-cylinder, resembling in other respects a non-medullated sympathetic nerve. Twenty-eight days after union these fibres have neither axis-cylinder nor medullated sheaths, yet are able to conduct mechanical and electrical stimuli

to the central nervous system. Fifty-eight days after union the nerve contains well-formed medullated fibres, which increase till about 88 days afterwards. At this stage a transverse section reveals well-marked bundles of small medullated fibres, between which are considerable gaps. In about 250 days the gaps are filled with larger fibres possessing nodes of Ranvier, and exhibiting the typical appearance of medullated fibres.

The process of regeneration of an afferent nerve which has been completely isolated for more than four weeks may be divided into three stages :—

1. The elongated spindle-shaped cells grow rapidly, and form fibres resembling the non-medullated sympathetic. In this condition they can transmit impulses four weeks after union with the normal median.

2. The fibres become medullated, and produce axis-cylinders within 58 days of union. Most are medullated at 88 days, but even after 200 days the nerve may consist of well-formed but small medullated fibres.

3. Larger fibres appear about 250 days after union. These stain deeply by Weigert's method, and are much larger.

PERCY T. HERRING.

## CLINICAL NEUROLOGY.

**RECKLINGHAUSEN'S DISEASE, WITH PIGMENTATION OF**  
(185) **THE MUCOUS MEMBRANES.** (*Maladie de Recklinghausen,*  
*avec pigmentation des muqueuses.*) ODDO, *Rev. Neurolog.*,  
1905, No. 8, April 30, p. 412.

Two typical cases of neurofibromatosis presented in addition pigmentation of certain of the mucous membranes—the glans penis and the lower lip in one case, the buccal mucous membrane and the inner surface of the cheek on the other. A possible relation between neurofibromatosis and Addison's disease may be explained from three points of view :—

- (a) That Recklinghausen's disease is itself due to pathological alteration in the suprarenals (Revilliod).

- (b) Or may exist coincidently with Addison's disease (Chauffard, etc.).

- (c) Or that involvement of the suprarenal plexus by neurofibromatosis gives rise to symptoms of Addison's disease (Jollin).

The author inclines to the last hypothesis.

S. A. K. WILSON.

**WESTPHAL-STRÜMPPELL'S DISEASE. WESTPHAL TYPE, SO-**  
**(186) CALLED PSEUDO-SCLEROSIS, AND STRÜMPPELL TYPE,**  
**SO-CALLED DIFFUSE SCLEROSIS.** (La malattia di West-  
 phal-Strümpell, tipo Westphal, cioè la così detta pseudosclerosi,  
 e tipo Strümpell, la così detta sclerosi diffusa.) R. REBIZZI,  
*Rivista di patologia nervosa e mentale*, Feb. 1905, p. 57, and March  
 1905, p. 105.

THIS is a very long and exhaustive paper on the subject of diffuse sclerosis and its connection with pseudo-sclerosis.

Rebizzi commences with a description of the clinical and pathological features of a case of diffuse sclerosis. The patient was a woman aged 31 years at the time of death. The first symptoms appeared at the age of 28, at the sixth month of her third pregnancy, with sudden transient weakness of the left side. This passed off, but was followed by gradual and progressive weakness and tremor of the left side, slight dysarthria, occasional incontinence of bladder and rectum, and a slight degree of psychical dulness. At this stage the patient went through a normal parturition. Afterwards the tremor and paresis of the left side increased, there was transient paresis of convergence, and about a year from the commencement of the symptoms the patient had a slight degree of mental feebleness, left-sided hemiparesis with intentional tremor, nystagmus, dilatation and immobility of the right pupil, and optic atrophy, more marked in the left eye. All the limbs, except the right upper limb, then became more spastic. Alternate remissions and relapses occurred. At last, after 2½ years, the patient became sleepless and noisy, and was admitted to the psychiatric clinic. On admission there was general spasticity of all the muscles, the lower limbs being flexed and the trunk slightly curved forwards. The patient was unable to stand or walk without support. All the limbs, except the right upper extremity, were weak and spastic, and showed intentional tremors. The right upper limb was practically normal. The deep reflexes in the right upper limb were brisk, in the left feeble. Both knee-jerks were absent, but there was double ankle-clonus and a bilateral extensor plantar reflex. Horizontal nystagmus was present, also mydriasis and immobility of the right pupil. Asymmetrical optic atrophy was present. Articulation was impaired. There was a moderate degree of simple dementia, with paroxysms of anger, weeping, and spasmodic laughter. Bedsores developed, and the patient died five months after admission, after an illness of about three years' duration.

The patient was born of syphilitic parents. She herself was married to a healthy husband at the age of 23. Her first two

children died in infancy, one from syphilis said to have been contracted from a wet-nurse. The last child, born during patient's illness, is alive and healthy.

At the autopsy the membranes and vessels were healthy, but there was marked increase in the consistence of the brain, which was abnormally firm on palpation. Its naked-eye slices, without artificial hardening, did not fall away or become rounded at the edges. No islands of disseminated sclerosis were present. Microscopically there was a diffuse, regular proliferation of the neuroglia all through the brain, more intense towards the surface. With this there was slight, diffuse disappearance of nerve-cells, especially those of the cortex and basal ganglia, also disappearance of certain fibres, especially the tangential fibres of the cortex. In the cord the pyramidal tracts and posterior columns had also a diminution of their fibres, though not well marked. The layer of neuroglia at the periphery of the cord was about three times the normal thickness. The spinal ganglia showed numerous "cell-colonies" of the type described by Sibelius. Such cell colonies, found originally in the spinal ganglia of syphilitic foetuses, are probably an indication of arrested development. Rebizzi regards their presence in the adult in this case as evidence of hereditary syphilis.

He then proceeds to discuss at great length the significance of the diffuse overgrowth of neuroglia in such cases. He maintains that the proliferation of neuroglia is not the primary phenomenon, but occurs secondarily to a prior destruction of nerve-elements. The morbid agent, whatever it be, destroys the nerve-cells and at the same time stimulates the neuroglia to proliferate. The excessive affection of the surface of the central nervous organ points, he thinks, to entrance of the morbid agent through the surface.

The symptoms of pseudo-sclerosis do not differ, save in degree, from those of diffuse sclerosis. In both forms, syphilitic or other toxic antecedents are common. Sex has no influence. The symptoms commence in childhood or in adult life, and the disease lasts from one to ten years. Remissions are more marked in the pseudo-sclerosis form than in the diffuse sclerosis. In both we observe progressive dementia, paroxysms of mental excitement, spasmodic laughter and weeping, syncopal and vertiginous attacks. Vomiting occurs in the diffuse sclerosis, but is rare in the pseudo-sclerosis type. In both the articulation becomes scanning, and ultimately incomprehensible. Slight unilateral facial weakness is common. Nystagmus, pupillary inequality and rigidity, are common in the diffuse type, rare in the pseudo-form. All voluntary movements become slow, and spastic paralyses with contractures develop in the limbs, generally more marked on one side. The deep reflexes are increased, often with ankle-clonus. Muscle-

tonus is exaggerated, and there is marked rigidity. There is also intentional tremor. Clonic spasms are less common in the diffuse variety than in pseudo-sclerosis. Acne and comedones are common in both. Pains and paræsthesiæ occur, and usually there is a general hypæsthesia and hypalgesia. The bladder and rectum are commonly affected in the diffuse type, rarely in the other. Bed-sores are rare in pseudo-sclerosis, not uncommon in diffuse sclerosis. Both varieties end fatally. Optic atrophy is a constant feature in the diffuse variety, whilst in the less severe type it is absent.

In cases of pseudo-sclerosis, no pathological changes have been found post-mortem, though Rebizzi is inclined to think that the pathological examination in such cases has not always been sufficiently exhaustive. In diffuse sclerosis, on the other hand, both naked-eye examination and microscopy have shown excessive density of neuroglia, the brain being sometimes of a semi-cartilaginous hardness. It is probable that many of the cases described as infantile disseminated sclerosis are really cases of diffuse sclerosis.

Rebizzi reminds the reader that the absence of microscopic changes on pathological examination does not exclude severe organic disease, and he cites the cases of other well-marked diseases without morbid anatomy, *e.g.* chronic chorea, paralysis agitans, and some forms of athetosis. He therefore places pseudo-sclerosis and diffuse sclerosis together in the same category, regarding them as two types of a disease essentially the same. They resemble certain cases of paralytic dementia in early life, and it is possible that certain cases recorded, without autopsy, as examples of juvenile general paralysis may be Westphal-Strümpell's disease. Syphilis as an etiological factor is present in both.

Clinically, the diagnosis between diffuse sclerosis and pseudo-sclerosis is extremely difficult. Only two signs seem to differentiate the two: the presence of pupillary changes and of optic atrophy in diffuse sclerosis and their absence in the other form; though even pupillary changes are not constantly absent in pseudo-sclerosis, and transient amblyopia has been observed. It is therefore practically impossible to say where pseudo-sclerosis stops and diffuse sclerosis begins. Moreover, there is no proportion between the intensity of the sclerosis and the severity of the symptoms.

Rebizzi regards the starting-point of the disease as essentially in the nerve-elements, not primarily a neuroglial overgrowth. For a discussion of his arguments the original paper should be consulted.

PURVES STEWART.

**COMBINED PSEUDO-SYSTEMIC DISEASE, WITH SPECIAL  
(187) REFERENCE TO ANNULAR DEGENERATION. A. R.  
ALLEN, *Univ. of Penn. Med. Bull.*, Jan. 1905, p. 382.**

At the age of 21, Mary L., of good family history, was taken ill with sickness. This continued for ten weeks, when she was brought up to the hospital unable to walk. Pain in the legs and trouble in walking came on six months previously, and for three weeks she had been unable to feel in the legs, and there had been occasional incontinence of urine.

On admission the legs were somewhat wasted, and showed general spasticity. There was anæsthesia to touch in the upper third of the thigh, but no anæsthesia to deep pressure or to cold. Heat anæsthesia was complete in the legs. There were no signs of vertebral disease.

The patient gradually became worse, developing facial paralysis and bed-sores. Screaming attacks and mental change preceded death, which occurred on July 12. No positive or negative mention of syphilis is made, nor is it stated whether the patient was married.

At the autopsy, acute miliary tuberculosis was found in the upper lobe of the left lung. Hæmorrhagic cystitis was present. No naked-eye description of the central nervous system is given. Sections from the lumbar region showed pyramidal degeneration, and some degeneration at the edge of the cord in the antero-lateral region. There was an intense infiltration of the pia by mononuclear round cells, and also some infiltration around the vessels in the cord.

In the thoracic region, sections show a zone of intense degeneration extending around the entire periphery of the cord. The columns of Goll are not degenerated in the lower parts of the dorsal region, but show marked degeneration in the upper regions. In the cervical region the same intense ring of degeneration around the edge of the cord was present. "This is probably caused by the thickening of the blood-vessels extending from the periphery of the cord inward. . . . It is very likely that the degeneration along the periphery of the cord was the result, in part at least, of toxic substances conveyed by the arteria corona."

Round-celled infiltration was also found around the vessels of the base of the brain, etc.

The author concludes that the case is one of intense meningo-encephalo-myelitis, probably of luetic origin. He thinks that the apparent systemic degeneration is produced by disease in the blood-vessels of the cord, particularly in the thoracic region.

STANLEY BARNES.



**MYELOMALACIA, WITH ESPECIAL REFERENCE TO DIAG-  
(188) NOSIS AND TREATMENT.** F. W. LANGDON, *Journ. of Nerv.  
and Ment. Dis.*, April 1905, p. 233.

THIS is a clear and concise account of the clinical differences between myelomalacia or acute thrombotic softening of the cord, and true inflammatory acute myelitis. Langdon also gives notes of two cases of myelomalacia and one of myelitis, the latter confirmed by autopsy.

In myelomalacia of thrombotic origin, a condition most commonly met with in arterial disease, syphilitic or otherwise, there is usually no preceding disability, the onset of paralysis is sudden, though there may have been premonitory transient numbness or other subjective sensations. Fever is absent and the tension of the pulse is not increased. There is no initial rigor. Rigidity of the spine is absent, likewise spasm of the extremities. The area of cord affected is often unilateral, and almost always asymmetrical. The paralysis, therefore, is often monoplegic. It extends by sudden increments. Dissociated sensory impairment is not uncommon, tending to the Brown-Sequard type. Girdle sensation is often absent. The knee-jerks are unlike, often unilaterally abolished, and the plantar reflexes are also unlike. A unilateral extensor response is common. The sphincters may be unaffected or may be uncontrolled for a few days only. Bed-sores are usually absent. There is no leucocytosis.

Inflammatory myelitis, on the other hand, usually occurs in patients the subjects of some preceding illness, injury, or acute infective process. The onset of paralysis is not sudden, but usually gradual. Fever is present and the tension of the pulse is high early in the disease. Rigor is not uncommon. Spinal rigidity and spasm of the extremities are frequent. The paralysis is always paraplegic in type and the extension of the process is steadily progressive. No dissociated sensory phenomena are found, the anæsthesia being bilateral and symmetrical below the lesion. Girdle sensation is present. The knee-jerks are abolished at the start, symmetrically, but later become exaggerated, if the lumbar enlargement have escaped. The plantar reflexes at first are abolished, but later become of the extensor type. The sphincters are severely affected, bed-sores are particularly common, and leucocytosis is probably present from the outset.

PURVES STEWART.

**THE CLINICAL CONNECTION OF BLINDNESS WITH TABES**  
 (189) **AND GENERAL PARALYSIS.** (*Relations cliniques de la*  
*cécité avec la paralysie générale et le tabes.*) LÉRI, *Journ. de*  
*Neurol.*, April 5, 1905, p. 121.

THE views of the writer harmonise with generally received opinion that blindness is rare in advanced general paralysis, while minor lesions of the visual apparatus are not; but some degree of amaurosis is not uncommonly a premonitory symptom of the disease. It may be remarked similarly that in advanced tabes, optic atrophy rarely supervenes, while minor disturbances of vision are not infrequent: but blindness frequently sets in before the ordinary phenomena of tabes are well established.

Hence it is maintained that optic atrophy has the same relation to general paralysis that it has to tabes: it is a rare complication of either, but it is frequently the precursor of them, occurring with minor tabetic and paralytic indications. It is possible that the reason for this is the same reason that an advanced tabes is rarely complicated with general paralysis. An ataxic is not more likely to become insane than blind. It is obviously undesirable to dogmatise on these relationships, but the author believes that tabetic amaurosis (so called) is a special localisation of a morbid process which may develop in two other ways, viz. in the spinal cord, clinically tabes, or in the cortex, clinically general paralysis of the insane.

S. A. K. WILSON.

**A CASE OF SPASTIC PARAPLEGIA WITH FOCAL SPINAL**  
 (190) **LESIONS WITH NO SECONDARY ASCENDING OR DE-**  
**SCENDING DEGENERATION.** (*Un cas de paraplégie spas-*  
*modique avec lésions médullaires en foyer sans dégénérescences*  
*apparentes dans la moelle ni au-dessus ni au-dessous de la*  
*lésion.*) GAUCKLER and ROUSSY, *Rev. Neurolog.*, No. 8, April 30,  
 1905, p. 409.

IN a typical case of spastic paraplegia the cord post-mortem was found at the level of the fourth and fifth dorsal segments to be very much reduced in volume, and to present on section a remarkably homogeneous appearance, it being impossible to determine the limits of the grey matter. Under a high power the section revealed a dense neuroglial sclerosis with the whorl arrangement in the posterior columns described in some cases of Friedreich's disease; scattered throughout were innumerable fine blood-vessels. At the same levels the anterior horn cells were conspicuously reduced in number and size. Nevertheless, at levels not more than two millimetres above and below this area of pathological alteration,

the cord appeared both to naked eye and to microscopical examination entirely normal. No trace of any ascending or descending degeneration was discovered by any of the various methods employed. The lesion being considered as a focal parenchymatous myelitis, it is possible that the abundant blood supply prevented degeneration from malnutrition, or that the cells were compressed rather than obliterated, or that the degenerated fibres, if there were any, were too scattered to be detected by Pal's method, or too old to render a Marchi examination positive.

S. A. K. WILSON.

**CONGENITAL SPASTIC RIGIDITY OF THE LIMBS. (CON-  
(191) GENITAL HYPERTONIA, LITTLE'S DISEASE.)** W. G.  
SPILLER, *Univ. of Penn. Med. Bull.*, Jan. 1905, p. 347.

IN the earlier part of this paper the author discusses at some length the clinical varieties of congenital spastic rigidity, pointing out that Little confused several types of conditions under one head. He then gives a résumé of four cases, two of which have already been reported, in which an autopsy occurred.

In the first case the outstanding feature was an unusual fineness of the fibres of the crossed pyramidal tracts, which Spiller considered to be evidence of arrested development. In the second case there were no marked signs in the spinal cord, the pyramidal tracts being well developed, but the clinical characters did not clearly define this as a case of spasticity. In the third case the anatomical changes found were similar to those in the first case. In the last case, the patient dying at the age of 70 after a lifetime (as far as could be ascertained) of spasticity of all four extremities associated with speech difficulty, there was found a compression of the upper cervical region of the cervical part of the spinal cord due to a partial dislocation of the vertebræ (? occurring at birth).

In reference to the pathology of this condition, we are surprised that no reference is made to the work of Collier, when many works of minor importance upon this subject are mentioned.

STANLEY BARNES.

**A CASE OF AMAUROTIC FAMILY IDIOCY.** JAMES BURNET,  
(192) *Journ. of Ment. Sc.*, Jan. 1905, p. 125.

THE child was a male, aged 18 months, parents Jews. Seventh child. Complaint, backwardness. He was apathetic, his eyes vacant, his tongue constantly protruded. He was rachitic, but no marks of degeneration were present about the body. He could not stand without support, nor sit up. Sensory functions seemed

normal, but he was slightly deaf and very blind, though he would look in the direction of a bright light. The optic discs appeared atrophied. He died of exhaustion after an attack of bronchopneumonia at 21 months. No post-mortem was obtained.

The family history is interesting. Child 1 died at 3 weeks; 2 became blind at 18 months, remained so for 18 months, and then recovered her sight after an attack of measles; 3 healthy; 4 became gradually blind and died at 13 months (of meningitis?); 5 healthy; 6 died of fits at 6 weeks; 7 the patient.

W. B. DRUMMOND.

**"SOUL PARALYSIS."** H. H. HOPPE, *Journ. of Nerv. and Ment. Dis.*, (193) March 1905, p. 146.

IN 1886 Munk first used the term "soul paralysis," to denote a form of paralysis produced by him in dogs by extirpation of the sensory cortical area. Hoppe records a clinical case of a similar kind—a loss of voluntary and spontaneous motor power in a case with motor centres and motor tracts intact, but presenting loss of sensation, central in origin, in the affected extremities.

His case is that of a woman, aged 47, who was seen four weeks after an apoplectic seizure followed by right hemiplegia. She had verbal amnesia, alexia, agraphia, often marked paraphasia; no hemianopsia. There was marked sensory loss in the right limbs—analgesia, thermo-anæsthesia, loss of muscle sense and sense of position, astereognosis; she did not know where right arm and leg were, unless she could see them. The right arm was completely paralysed, quite flaccid, reflexes were absent: she could not make the slightest spontaneous movement with it, but, if she was told to watch the doctor's hand closely, and then told to repeat the movement, the movement could be executed at once. She could move the right leg slightly, but had made no attempt to walk; the muscles were relaxed, the foot in typical condition of foot-drop, knee-jerk was present, Babinski's sign positive. She regained the power of making gross movements of the arm when asked to do so, but, even after eight months, the right hand and arm were used spontaneously only on the rarest occasions and by a special effort of the will; all movements were less perfect when performed with closed eyes; the finer movements were very imperfect; she was unable to imitate with the normal arm any passive movements made on the paralysed arm, but was able to imitate all movements of the normal arm with the paralysed arm; the muscular power was almost normal, the sensory loss still very marked. The leg had improved in power, but showed phenomena very similar to those in the arm.

Similar cases in the literature are referred to shortly. The pathological condition underlying this condition is "a destruction of the cortex or the subcortical region of the parietal or sphenotemporal lobes, or of the sensory tracts."

Soul paralysis has many points of resemblance to soul blindness, and also to amnesic aphasia. The condition is explained thus:—A spontaneous or voluntary act is possible only if the higher cortical reflex arc is intact; "the fineness, precision, and, in fact, very often the origin of voluntary muscular movements are dependent upon preceding sensory impressions and knowledge which are, unconsciously, utilised in each movement which is made: if, therefore, the motor centre and motor tracts are intact, but the sensory centres or association fibres connecting the sensory centres with the motor tracts are destroyed, the sensory irritations for muscular movements no longer reach the motor cortical area; there is a break in the higher cortical reflex arc, and a paralysis results, not due to a break in the motor section of this reflex arc, but in the sensory." The senses, whose loss is the chief factor in the production of soul paralysis, are the muscle sense and sense of position. Cases, in which all the qualities of sensation are interfered with and yet no soul paralysis results, are explained thus:—Soul paralysis requires for its production a loss of *all* association impulses from the sensory centres—there must be a destruction of the association tracts connecting these centres with the higher psychic centres as well as with the motor centres. Again, while the patient may be unable to make the slightest movement spontaneously, he may be able to do so when the hand is seen, or when he is commanded to make the movement, or when he imitates passive movement of the sound arm with the paralysed arm: this is due to the fact that tracts from the visual and auditory centres and from the sensory centres of the opposite side to the motor area of the apparently paralysed side are intact.

Hoppe also discusses the bearing of experimental work, notably that of Mott and Sherrington and Munk, on the subject of the relation of sensation to motion.

A. W. MACKINTOSH.

**THE CEREBELLAR SEIZURE (CEREBELLAR FITS). A SYN-  
(194) DROME CHARACTERISTIC OF CEREBELLAR TUMOURS.**

CHARLES L. DANA, *New York Med. Journ.*, Feb. 1905.

THIS paper contains the report of a case of tumour situated in the cerebello-pontine angle. The fits which occurred in this case are more specially described. At first they consisted of "rushes of blood to the head," dizziness and roaring in the head with inability

to stand, but without loss of consciousness. These fits lasted from five to thirty minutes and generally occurred during the day. Later they became more severe and were accompanied by loss of consciousness and stiffening and irregular tonic movements of the limbs. The author describes these attacks as cerebellar or cerebello-pontile seizures. The special syndrome of cerebellar and posterior fossa tumours is described thus: (1) Loud, high-pitched tinnitus or roaring and crackling noises, rapidly increased in intensity. (2) Vertigo, usually objective, and with or without forced movements. (3) A tendency to drop or fall in one direction or another instantly to the ground. (4) Sometimes sudden blindness and loss of consciousness. (5) In severe attacks, tonic spasms generally of an extensor type. This lasts from one to two to five or ten minutes.

T. GRAINGER STEWART.

**RECURRENT OCULO-MOTOR PALSY, WITH REPORT OF A**  
(195) **CASE.** W. G. SPILLER and W. C. POSEY, *American Journ. of Med. Sc.*, April 1905, p. 587.

THERE are not on record a great many cases of recurrent oculo-motor paralysis; the authors contribute another to the list. It is that of a man of thirty-one, who had had vague symptoms for about five years. His condition at the time of examination was entirely normal, save for the fact that his right third nerve was completely paralysed, except as regards the interior muscles of the eye. His family history revealed nothing noteworthy, and in regard to his personal history, the only point of interest was the history of this condition; syphilis was excluded. At the age of fourteen or fifteen he had been very subject to attacks of migraine, in which he became temporarily blind, except that he still retained light perception. Such an attack would last for half an hour, more or less, and then would ensue a violent headache, during which flashes of light annoyed him; the "blindness" was always gone before the headache came on. These attacks gradually passed off, and he had had none for ten years. In January 1904 he had transitory diplopia, in which the double vision was chiefly to be made out when he looked upwards and inwards. There was no ptosis. This seizure was preceded by severe pain in the eye, and had apparently been determined by overwork. It passed away after several weeks, but returned again in July, accompanied by ptosis, as above mentioned. After four months the ptosis became less, but did not quite disappear. The authors compare their case with certain of those already published, and discuss the explanation given by Möbius of the cause of such an occurrence, that it is a basal lesion. They regard it, not as a disease, but as a symptom-complex.

W. G. SYM.

**ON HYSTERICAL MONOCULAR AMBLYOPIA AND ITS  
(196) DISAPPEARANCE IN BINOCULAR VISION (A PROPOS  
OF A NEW CASE). (Sur l'amblyopie hystérique monoculaire  
et sa disparition dans la vision binoculaire.) R. GRUGHET,  
*Archiv. de Neurolog.*, May 1905, p. 337.**

THE case cited is that of a girl æt. 14½ years, who was admitted into hospital suffering from right hemianæsthesia and amblyopia of the right eye. The girl's mother was a highly nervous woman, and the father had died in an attack of delirium tremens. The child had always been very timid, and had suffered much from night terrors.

The first symptoms of eye trouble began about one and a half years previously, when the girl suddenly felt a painful sensation in the right eye just as if she had a grain of sand in it. Some injection of the eye followed, and headache was complained of. A slight phlyctenular keratitis developed, and though the eye recovered under appropriate treatment, certain functional troubles became prominent, *e.g.* headache, intermittent diplopia, micropsia, when objects were looked at with the right eye only. About nine months after the onset of eye symptoms dilatation of the right pupil was noticed along with amblyopia of the right eye. The amblyopia was not absolute, the patient reading four or five lines of Snellen's types. The letters on the scale appeared greatly diminished in size. The fundus oculi was perfectly normal. The mydriasis varied greatly in amount, and finally passed off in about a month, but the amblyopia persisted. A right-sided hemianæsthesia then developed; the symptoms were diagnosed as hysterical, and electrical treatment was tried. Hiccough, accompanied by a sort of globus hystericus, and a hysterical fit in which the patient sang and laughed by turns, tore her hair, and lay kicking on the ground, were subsequently noted; also a spasmodic rotation of the head to the right, winking of the eyes, frowning, and shrugging of the shoulders.

On admission into hospital the right hemianæsthesia was found to be complete for contact, pain, and temperature. Electric cutaneous sensibility was not completely abolished. The osseous, tendinous, and nervous sensibility of the deeper parts was abolished, while the muscular and stereognostic senses were preserved. The ovarian, plantar, deep epigastric, tracheal, and pharyngeal reflexes were absent on the right side. Movements were executed perfectly, but the grasp of the right hand was slightly weakened. Taste and smell were normal, hearing slightly diminished on the right side.

In the eyes the conjunctiva and cornea of the right side were insensitive, but touching the cornea provoked a flow of tears. The pupils were equal and reacted normally, except that when the sound eye was closed the right pupil was larger than was the left pupil when the right eye was closed. In the right eye hyperm. astig. 2 D, in the left eye .75 D, L.V. =  $\frac{2}{3}$ . With right eye patient said she saw nothing. She, however, could distinguish objects brought near the eye. A black circle on a white card was used as a test object, and marked micropsia was found to exist. No diplopia nor polyopia; accommodation of left eye = 14 D, of right eye = 5.5 D. Chromatic sense of right eye perverted, *e.g.* sky-blue seen as white, deep reds or greens seen black. The field was somewhat restricted on both sides.

Finally, in spite of the apparent amblyopia of the right eye, binocular vision was found to be present, the amblyopia disappearing when both eyes were open. A large number of experiments were made proving the existence of binocular vision, and also of good monocular vision in the right eye when acting independently, *e.g.* with a prism before one eye the patient saw double, and the images were of equal size. Similarly the dyschromatopsia was shown to be absent when both eyes were open. A pencil or ruler held between her eyes and a printed page did not prevent her reading any of the words, showing that both eyes were used. Among other tests employed were Snellen's well-known test with red and green letters, various tests with the stereoscope, etc., etc.

Certain of the experiments were so planned as to prove the existence of independent monocular vision in the apparently amblyopic right eye.

Closure of the sound eye by the lids or by complete occlusion called forth the amblyopia of the other eye. Merely screening the sound eye without preventing the entrance of light had not this effect. The chief conclusions arrived at regarding this case may be summarised as follows:—

- (1) The amblyopia disappeared in binocular vision.
- (2) The amblyopic eye could be proved to see not only binocularly but monocularly and independently.
- (3) The disappearance of the amblyopia was not explained by the exercise of binocular vision, nor by the simultaneous exercise of monocular vision in each eye. The amblyopia appeared when the sound eye was closed, and disappeared when it was open.
- (5) Closure or occlusion of the healthy eye acts by cutting off the light stimulus.

J. V. PATERSON.



**MYASTHENIA GRAVIS, WITH PARALYSIS CONFINED TO**  
**(197) THE OCULAR MUSCLES.** W. G. SPILLER and ERNEST N.  
 BUCHMAN, *Am. Journ. of Medical Sciences*, April 1905, p. 593.

MAN, æt. 33, complained of dizziness and blurring of sight of one week's duration. The vision was about normal, though slight error of refraction existed. Six months later complete paralysis of the right inferior rectus was present, and soon afterwards ptosis of the left eye, occasionally of the right. A diplopia existed, caused by partial paralysis of the internal rectus and complete paralysis of the inferior rectus of the right eye. On rising in the morning the eyes were wide open and the double images very close together, and at times he had no perceptible diplopia, but as he resumed the duties of the day all the symptoms recurred. A later note reads:—When he takes off his glasses the upper lid of one eye, depending on which eye he had been using last, begins to fall, and gradually the upper lid falls until there is complete ptosis; while this is occurring the upper lid of the other eye gradually drops until ptosis may be complete or nearly complete on this side.

There were no other symptoms beyond a suggestion of the myasthenic reaction in the sternocleidomastoid muscles.

The writers allude to the rarity in which the myasthenic condition is limited to the ocular muscles, and point out that it may be confused with the ocular pareses of tabes.

They mention Sterling's case in which there was a ptosis of varying intensity, and also the myasthenic reaction in the biceps, deltoid, and sternomastoid muscles. Oppenheim has recorded another case in which later bulbar symptoms appeared and which terminated fatally.

W. B. WARRINGTON.

**SOME NEW BONE REFLEXES OF THE LOWER EXTREMITIES**  
**(198) UNDER NORMAL AND UNDER PATHOLOGICAL CON-**  
**DITIONS.** (Ueber einige neue Knochenreflexe der unteren  
 Gliedmaassen im gesunden und im pathologischen Zustande).  
 VALOBRA and BERTOLOTTI, *Neurolog. Centralb.*, April 16, 1905,  
 p. 343.

1. On tapping the internal malleolus, the subject lying on his back and the foot slightly everted, one gets, in 35 per cent. of cases (normal individuals, or, at least, without any evidence of disease of the nervous system), a contraction of the adductors of the thigh on the same side.

2. On tapping the plantar aspect of the heel, the leg being

extended and stretched over the bed, in 40 per cent. of cases the adductors on the opposite side contract.

3. On percussing the internal condyle of the femur, or the head of the tibia or its shaft, the legs being flexed at the knee and slightly apart, in 60 per cent. of cases there is contraction of the adductors on the same side; in 50 per cent. on both sides.

4. Percussion of the patella, the patient being on his back and the knee flexed, produces contraction of the thigh adductors on the opposite side in 50 per cent. of cases.

It is essential in these examinations to have a heavy hammer, which will give a smart blow without producing any pain.

The authors consider that the production of the reflex in the opposite limb depends to a great extent on the position of the limb which is percussed. In several of their cases they have been able to observe a dissociation between these bone reflexes and the tendon reflexes in the same limb. In sciatica, tabes, multiple neuritis, they have found, *e.g.*, the Achilles-jerk absent, but the bone reflexes present.

In a case of complete transverse lesion of the cord at the level of the eleventh dorsal segment, all cutaneous sensibility, all appreciation of passive movement were lost; nevertheless, percussion of the bones in their subcutaneous areas was very painful. Possibly, the percussion of these structures produces a purely mechanical vibration which acts as a stimulus to posterior nerve roots. It is possible that some such stimulus as this excites a reflex action from posterior roots to anterior horn cells. Why the way out should be preferably *via* the adductors is not explained.

S. A. K. WILSON.

## PSYCHIATRY.

### ON THE TECHNIQUE OF CLINICAL PSYCHOLOGICAL EXAMINATION.

(199) NATION. (Zur klinisch-psychologischen Untersuchungstechnik.) K. HEILBRONNER (of Utrecht), *Monatsschr. f. Psych. u. Neur.*, Feb. 1905.

HEILBRONNER calls attention to a method of examination which has given him useful results in his cases. He had found that a patient with a typhoid psychosis grasped the meaning of a picture and could compare it with a second picture, while a patient with an eclamptic psychosis did not grasp the picture as a whole, but named correctly objects in the picture in great detail. The question arose whether the patient could not grasp the picture owing to inability to combine the numerous partial impressions, or owing to inability to receive more than a very limited number of im-

pressions at the same time. To eliminate the second factor, Heilbronner used a very simple series of drawings of common objects. The first drawing was a bare outline, in the next a few lines were added, and so on until in the last drawing of the series the object was represented clearly and with some detail. It was found that the patient with the eclamptic psychosis was able always to point out what lines or dots had been added in the successive drawings; her inability to correctly name the pictures was due to inability to combine the numerous partial impressions.

The use of this method enables one to determine not only the combining ability, but also the perception of the patient. Heilbronner gives a short account of the results obtained in stuporous conditions, and in defect conditions whether congenital or the result of a psychosis. Stuporous patients gave the worst results, imbeciles were able to recognise the drawings only in their final or almost complete form, some paralytics gave very good results, hebephrenics varied considerably. Not only does the method enable one to estimate the patients' grasp, it is useful for eliciting flight of ideas where that symptom might otherwise be overlooked. The same simple test may be used to determine the reaction time of the patient, the results of fatigue, the condition of retention, and at the same time other symptoms, such as perseveration, are often well demonstrated.

C. MACFIE CAMPBELL.

**ON ENTOPTIC PERCEPTION OF THE CIRCULATION AND ITS  
(200) IMPORTANCE IN THE THEORY OF VISUAL HALLU-  
CINATIONS.** (Ueber entoptische Wahrnehmung des eigenen  
Blutkreislaufes, etc.) A. PICK, *Wien. klin. Wchnschr.*, Feb. 16,  
1905, p. 159.

PICK seeks to explain certain visual hallucinations, as based upon a false interpretation of sensations due to the circulation of blood in the retina. He refers to Meynert's view that the animal hallucinations in delirium tremens are due to the movement of the blood in the retina, causing scotomata which are taken for little animals. Helmholtz explained the little spots seen on gazing persistently at a uniformly illumined surface (Exner's experiment), as due to the passage of the blood corpuscles through the capillaries.

The author reports the observation of a patient 42 years of age, who, in 1897, twelve years after syphilitic infection, suffered from shooting pains, and one year later had epileptiform attacks with transitory aphasia. In 1898 he developed a psychosis which lasted for three months. In 1905 physical examination disclosed the following symptoms:—complete blindness, optic atrophy, Argyll-

Robertson pupil, absence of patellar and Achillis reflexes, slight hypotonia ; but no sign of Romberg.

Patient said that he saw in his eyes luminous objects : they were like little balls or threads, varying in colour from day to day ; they were in constant motion from the periphery to the centre, where they formed a regular whirlpool, and thence back to the periphery. Patient also compared the moving lights to an exceedingly fine spray. The phenomenon annoyed the patient so much that he requested the doctor to remove the eyes.

Pick calls attention to the similarity of the phenomenon, as described by the patient, with what is observed in Exner's experiment. He explains the occurrence of such visual perceptions of peripheral origin, in a person blind through optic atrophy, by the persistence of a certain number of fibres in the optic nerve and of a certain amount of functionally active nervous tissue in the retina. The exclusion of the ordinary optic sensations would give an abnormal prominence to sensations due to the impression of the circulation in the retina on the nerve tissue still functioning. The "entoptic" perception of the circulation of the blood must therefore be taken into consideration in discussing the visual hallucinations of tabetics.

C. MACFIE CAMPBELL.

**ON PSEUDO - MELANCHOLIA. (Ueber Pseudo - Melancholie.)**

(201) O. JULIUSBURGER (of Schlactensee), *Monatsschr. f. Psych. u. Neur.*, Jan. 1905.

THE author reports the case of a woman 30 years old, who for several years had the subjective symptoms of melancholia—depression, inability to form a decision, blocking of thought, ideas of unworthiness and of suicide. For a certain period she complained of inability to call up before her mind's eye familiar places and friends ; she lost the power of feeling joy or pain, love or longing, hunger or the feeling of repletion.

There were no objective signs of the subjective insufficiency, no trace of retardation, no want of spontaneous movements nor lack of facial expression ; perception, memory, and grasp were excellent ; her mood seemed uniformly placid. The author traces the symptoms to the loss of organic sensations, to somatopsychic afuction in the terminology of Wernicke ; in depressive melancholia (depressed stage of manic depressive insanity) we have in addition to objective signs the same somatopsychic afuction. On this ground Juliusburger considers "pseudo-melancholia" to be a more appropriate description of the case than "somatopsychosis" ; pseudo-melancholia has, however, been already used by Wernicke to describe a certain form of his "depressive melancholia."

C. MACFIE CAMPBELL.

**ON PSEUDO-MELANCHOLIC CONDITIONS.** (Ueber pseudomelancholische Zustände.) W. VORKASTNER (of Berlin), *Monatssch. f. Psych. u. Neur.*, Feb. 1905.

VORKASTNER reports the cases of eight patients who, in the course of their psychoses, presented a depressed stage with certain features of melancholia, which term he uses in rather a vague manner. In the first four cases the depression preceded the development of a paranoic condition; in the other cases an acute hallucinatory paranoia (Ziehen) passed into a condition of depression with ideas of sinfulness. The cases are very heterogeneous, and their discussion is rather too theoretical to be helpful clinically.

C. MACFIE CAMPBELL.

**CONSIDERATIONS ON DEMENTIA PRÆCOX.** (Considérations sur la démence précoce.) E. MARANDON DE MONTYEL (of Ville-Evrard), *Journ. de Neur.*, Jan. 5, 20, 1905.

THE author considers that everything in Kraepelin's conception of the deteriorating psychoses is false. There is a dementia præcox, beginning between the ages of 14 and 25 and leading quickly to dementia, but this true dementia præcox has nothing to do with that of Kraepelin. The latter belongs really to "mental confusion," and might be called chronic mental confusion, or in certain cases "post-confusional systematised paranoia" (paranoid form of dementia præcox). In criticising the term "dementia præcox" as used by Kraepelin, he calls attention to the number of cases that recover: Kraepelin has found that 8 per cent. of his hebephrenics, and 13 per cent. of his catatonics recovered, therefore, according to the author's method of calculation, 21 per cent. of cases of dementia præcox recover. An extension of this arithmetical method leads him to the conclusion that 65 per cent. of cases recover sufficiently to resume their occupations!

The article is theoretical, contains no clinical facts, and is an excellent proof of the confusion that is inevitable with a purely symptomatological grouping of the psychoses, and of the great advance Kraepelin made by insisting upon their course and outcome.

C. MACFIE CAMPBELL.

**ALLOPSYCHIC MANIAS.** (Allopsychische Manien.) KNAPP (of Halle), *Monatsschr. f. Psych. u. Neur.*, Jan. 1905, p. 56.

THE exaggerated feeling of ability of the manic, depending on his disordered mental activity, may lead to ideas of greatness with a

completely false conception of his own personality; occasionally hypochondriacal ideas develop on the basis of trivial complaints, which are felt to an exaggerated degree by the patient with his expansive ideas.

To use Wernicke's terminology, an alteration in the content of consciousness, both in the autopsychic and in the somatopsychic sphere, may result from the disordered activity of consciousness of the manic. Knapp gives the observation of a manic in whom there was a marked disorder in the allopsychic sphere. The patient presented the cardinal symptoms of mania, flight of ideas, elation, a great flow of speech and action, a general levelling of ideas; in his second attack the symptoms increased until patient presented the clinical picture of "confused mania" (Wernicke), with motor perplexity and irritative psychomotor symptoms. The interesting feature in the case was that in the first attack patient showed a marked paranoid trend, with false identification of other patients. In the second attack, with absolutely clear sensorium, patient mistook patients for old friends, the attendants for court officials, and was for a time disoriented as to place.

Knapp explains the paranoid trend as due to the conflict of the expansive personality of the manic with his environment; it therefore does not go beyond the manic symptom-complex. False identification of persons and disorientation, however, are usually referred either to the clouding of the sensorium, superficial observation, expansive feeling, or mere jesting, which occur in a manic attack. These did not explain the allopsychic symptoms in this case; the allopsychic symptoms were of independent origin, and therefore went beyond the ordinary symptoms of a pure manic attack. For such cases the author proposes the term of "allopsychic manias," rather than Wernicke's "maniacal allopsychoses," as the allopsychic symptoms were only accessory, and the allopsychic disorientation only partial.

C. MACFIE CAMPBELL.

#### ON THE DELIMITATION OF CHRONIC ALCOHOL-PARANOIA.

(205) (*Zur Abgrenzung der chronischen Alkohol-paranoia.*) RAECKE  
(of Kiel), *Arch. f. Psych. u. Nerven*., Bd. 39, H. 2.

RAECKE calls attention to the contrast between the agreement of most authors on the clinical forms of the acute alcoholic psychoses, and the confusion that exists with regard to the chronic alcoholic psychoses. He reviews the opinions of those who have written on this subject, and attributes the confusion in great part to the vague conceptions of the nature of "chronic paranoid conditions" and "delusions of jealousy" (*Eifersuchtswahn*).

Too much stress has been laid on delusions of jealousy, for the

development of which the marital relations of the alcoholic are very favourable. What is important is not so much the nature of these delusions, as their delusional foundation or elaboration. These delusions may be of extremely short duration and be quite forgotten later, if they are produced under the excitement of acute intoxication or a paroxysm of passion; they may be elements in a paranoid episode following repeated alcoholic excesses. Such paranoid episodes are usually accompanied by ill-humour, anxiety and unease, irritability, disturbance of sleep and appetite; they have nothing in common with chronic paranoia. When a series of such episodes occurs without the correction of the delusions in the intervals, we have a type of psychoses under which most cases of chronic paranoid alcoholic insanity come: such cases improve or remain stationary if the patients stop drinking, and thus are distinguished from true paranoia.

In some cases, however, we find a fixed delusional system which undergoes further elaboration, and is entitled to the name of "chronic alcohol-paranoia."

Raecke gives fully five typical cases of this group; the patients showed the development of a chronic psychosis similar in its nature and course to the classical paranoia; there was no marked heredity nor proof of a previous paranoic disposition. It seems, then, that chronic paranoia may develop on the basis of mental inferiority, either congenital or acquired, through prolonged alcoholic excesses. As to the frequency of this type, Raecke found only 3 cases in 200 cases of alcoholic psychosis. His conclusions are the following: 1. There is an alcoholic paranoia. This develops on the basis of chronic alcoholism either primarily or following immediately delirium tremens or acute hallucinatory insanity. 2. Clinically chronic alcoholic paranoia is almost identical with classical paranoia; the prognosis is unfavourable. 3. Chronic alcoholic paranoia is to be separated clearly from transitory paranoid excitements, and from the terminal conditions of deterioration which are seen after delirium tremens and acute hallucinatory insanity.

C. MACFIE CAMPBELL.

**CLINICAL AND ANATOMICAL STUDIES UPON JUVENILE**  
(206) **GENERAL PARALYSIS.** (*Studi Clinici ed Anatomico-patologici sulla Paralisi Generale Giovanile.*) BURZIO, *Annali di Freniatria*, March 1905, p. 33.

THIS article consists of notes of three cases.

CASE I. Man, 23 years. In infancy acquired syphilis from his mother. In disposition, solitary and taciturn, but industrious. About 8 months before admission to the asylum he took ill; he

had gastric disturbance and insomnia, became melancholic, had hallucinations of sight and hearing, fits of impulsiveness and excitement, epileptiform attacks, twitchings in muscles of face and right leg.

Upon examination he presented signs of incomplete development (childish expression, absence of hair on face). Knee-jerks exaggerated. Plantar reflex absent. Weakness in reaction of pupil to light; tremor of the tongue. He was at first depressed, later exalted and talkative. Intelligence and memory gradually became worse. Pupils were unequal in size. With affection of speech and grandiose ideas, he soon completed the classical picture of general paralysis. Later appeared paraplegic symptoms and bed-sores, giving rise to septicæmia, which soon carried him off, a little over two years after admission.

*P.M.*—Calvarium uniformly thickened, dura mater on left side thickened owing to a deposit of fibrin, no adhesions between pia arachnoid and brain substance, atrophy of the convolutions, especially in the frontal lobes, oedema of brain, faint granulations on the walls of the dilated lateral ventricles and on the floor of the fourth ventricle. Pachymeningitis of the cord, degeneration of the pyramidal tracts, of the columns of Goll and of the anterior and posterior nerve roots.

CASE II. Girl, 18; mother dead, cause unknown; father died in an asylum from general paralysis. Of poor intelligence, and almost unfitted for study. Some months before admission became sleepless, somewhat deranged, at times exalted and physically weak. Hands tremulous.

Clinically, head and face were poorly developed. General nutrition poor, expression childish, sexual organs scarcely at all developed (amenorrhœa). Pupils equal, dilated, reacted well to light, knee-jerks exaggerated, speech disjointed, memory weak and confused. Mentally exalted. A period of quiescence followed, with slight improvement, during which patient was able to do light manual labour. Later the dementia became more marked, the tremor increased, lower limbs became ataxic and progressively weaker. Attacks of Jacksonian epilepsy appeared on the right side, followed by hemiplegia on same side. Pupils became insensible to light. Speech became markedly affected. Trophic lesions in skin of thighs and heels. Patient died of advanced marasmus, little more than four years after the initial onset.

*P.M.*—Calvarium thin, light, almost transparent in places, diploe almost absent. Fibrinous clot in longitudinal sinus. Brain shows marked atrophy, affecting especially the frontal lobes and cortex. Very firm in consistence. Excess of fluid in lateral ventricles. Granulations on floor of fourth ventricle. Histologically there was absence of the tangential fibres, atrophy of



the ganglion cells, congestion, marked increase of the interstitial tissue. Similar sclerosis in spinal cord. Left lung showed atelectasis. Marked atrophy of the intestinal mucosa. Mucosa of uterus covered with abundant catarrhal effusion. This and the next case showed fatty changes in heart and liver.

CASE III. Man, 21. Personal and family history good. Good worker. In 16th year, while going to work, he took an ordinary epileptic fit. This was followed by paresis of muscles of tongue and pharynx. Two months later had a second and more serious fit. Consecutive fits were milder. Patient became taciturn and misanthropic. At times apathetic, at other times violent and impulsive.

Clinically, general nutrition poor, signs of arrested development, *e.g.* childish expression, absence of hair, etc. Knee-jerks lively. Pupil reaction slight. Patient often sleepless.

He commenced to have attacks of excitement, followed by unconsciousness, during which pulse was quickened and temperature rose ( $38.3^{\circ}$  C.). They came on every few days, and were accompanied by lockjaw, which became continuous. The pupils were dilated, barely reacting to light, and not at all to accommodation. Plantar reflex very weak. Babinski's sign present. Knee-jerks exaggerated, especially the right. Bed-sores appeared. Patient died about five years after onset of the disease.

*P.M.*—Meninges and blood-vessels normal. Well marked granulations in lateral ventricles and on floor of 4th ventricle. Brain substance normal in consistence, except in occipital lobes, where there was a zone of sclerosis about 1 sq. cm. in area lying in white matter of each lobe, with the same colour as surrounding tissue. There was a similar less extensive zone in the pons and medulla.

On microscopical examination, tangential fibres of cortex were almost normal. There was a slight amount of round cell infiltration of the walls of the small vessels and under pia mater. No noteworthy changes in spinal cord. Mucous membrane of intestine hyperæmic, with atrophy of the follicles.

In this case the symptoms were due to a circumscribed interstitial encephalitis.

In conclusion, the author remarks that these results show that juvenile general paralysis, although analogous to the adult form, is characterised by the frequency of signs of arrest of development at puberty, by the predominance of bodily phenomena (tremors, fits, etc.) over the mental disturbances (dementia with or without delusions), by the frequency with which the cerebral alterations are complicated by important lesions of the medulla and spinal cord, and by the variations in its pathological anatomy.

R. G. GORDON.

**TREATMENT.**

**SCLAVO'S IODISED GELATINE IN THE TREATMENT OF (207) EXOPHTHALMIC GOITRE.** (*La gelatina iodata Sclavo nella cura del morbo di Basedow.*) VALERIO LUSINI, *Riv. crit. di clin. med.*, 1905, Anno vi., p. 153.

LUSINI's paper deals with the therapeutic value of iodine-proteid compounds in the treatment of exophthalmic goitre.

In a former contribution he recorded a case of that disease cured five years ago by means of hypodermic injections of Sclavo's iodised milk. The patient still remains well; the thyroid has not undergone enlargement, exophthalmos has disappeared, the rate of the heart remains normal, and the patient has not been subject to any of the symptoms from which she suffered prior to treatment.

Lusini now records a case successfully treated by means of iodised gelatine. The patient, a girl of twenty years, who had previously always enjoyed good health, had noticed that the size of her neck had been increasing for some months, and complained of frequent palpitation. After an acute illness—intestinal auto-intoxication—there was rapid increase in the size of the thyroid, sensation of suffocation, vertigo, bilateral exophthalmos, and signs of cardiac dilatation, with a pulse-rate of 125 to 130 per minute, rising to 160 to 170 on excitement or slight exertion. No real benefit was obtained by administration of large doses of sodium and potassium iodide in combination with sodium bromide, by local application of iodine to the neck, nor by means of thyroïdin.

The administration of Sclavo's iodo-gelatine (2 grammes of iodine in 100 grammes of gelatine) was then commenced, the daily dose by the mouth being from three to four teaspoonfuls. Twenty days later the patient was sensible of improvement, her symptoms were less pronounced, the thyroid gland, though of unchanged size, had become softer, while the pulse-rate was 115 to 120 per minute. The iodo-gelatine was then given hypodermically, 5 c.c. (containing 0.10 gramme of iodine) being given daily for two periods, each of twelve days, with an interval of a week between them. Improvement commenced after the first injection, and after the second series of injections was completed the thyroid swelling had completely disappeared; the pain in the shoulder and neck, of which she had previously complained, the vertigo, palpitation and sensation of suffocation had all ceased; the pulse-rate had fallen to 74 to 78 per minute, and the cardiac impulse was reduced almost to its normal limits; the sole remaining sign of the disease being the exophthalmos. The patient gained markedly in weight, and has remained in excellent health since the treatment was stopped four months ago.

W. T. RITCHIE.

- THE IMPORTANCE AND THE DIFFICULTIES OF A "SODIUM-  
(208) CHLORIDE FREE" DIET AND THE TREATMENT OF  
EPILEPSY.** (Die Bedeutung und die Beschwerden der  
Kochsalzenthaltung und die Behandlung der Epilepsie.)  
L. J. J. MUSKENS, *Neurolog. Centralb.*, March 1, 1905, p. 208.

MUSKENS made observations on 180 cases of epilepsy which he treated by the method of hypochlorisation, introduced by Richet and Toulouse. The author is of opinion that the amount of sod. chloride present in the body may fluctuate very considerably without harmful results. Many individuals (especially males) can be absolutely deprived of NaCl ingestion provided equimolecular bromine salts are supplied. Some South American peoples dispense practically entirely with sod. chloride in their dietary.

Muskens finds that hypochlorisation intensifies the action of bromine salts on the pathological process in epilepsy and in causing acne. Grand mal is more easily influenced than petit mal or the psychical symptoms. Hypochlorisation may cause vertigo, disturbance of speech, loss of memory, constipation or diarrhoea, and stubborn trigeminal neuralgia in those who do not bear the treatment well. Heart disease, especially myocardial affections, is the only contra-indication to the treatment. The degree to which hypochlorisation may be pushed varies very much in different cases. It is found that by this method of treatment the bromine is much more slowly excreted from the body. The theory is that bromine has a greater affinity for certain metabolic products than chlorine has, and that the latter combination is the more toxic.

J. EASON.

- BILATERAL CERVICAL SYMPATHECTOMY FOR THE RELIEF  
(209) OF EPILEPSY, WITH REPORT OF THREE CASES; NOTES  
ON THE PHYSIOLOGIC EFFECTS OF CUTTING THE  
SYMPATHETIC, AND ON THE HISTOLOGIC CHANGES  
FOUND IN THE CASES IN QUESTION.** W. P. SPRATLING  
and R. PARK, *Journ. of Nerv. and Ment. Dis.*, April 1905,  
p. 218.

THIS paper, as its title indicates, records the treatment of three cases of epilepsy by bilateral excision of the sympathetic chain in the neck. They were all operated on by Dr Park on the same date, November 16, 1903. Of the first case it is stated that he made a "rapid and satisfactory recovery from the operation." Nothing is said as to the effect on the epilepsy. Of the second case no bulletins are given after the day following operation, whilst

in the third all we learn is that the double ptosis which resulted passed off in about two weeks. No notes, curiously enough, appear as to the subsequent course of the epilepsy, though in one case the writers remark that "an additional condition, a pronounced 'tic' of the head and right arm, was radically improved in a way that seemed a year later would be lasting."

The value of these cases, therefore, is entirely negative as regards the therapeutic effect of the operation. Most neurologists are familiar with Jonnesco's series of operations, published in 1902, in which cure was only claimed in four cases out of ninety-six. Nor do cases like these of Spratling and Park, devoid of subsequent history, throw any additional light on the subject.

The paper also records some of the clinical appearances of sympathetic palsy in man. The histological appearances in the sympathetic itself are practically normal, as one would expect. The presence of a double nucleus in some of the nerve cells is called attention to.

PURVES STEWART.

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## Original Articles

### **SOME OBSERVATIONS ON THE EFFECTS PRODUCED BY CHOLINE UPON ANIMALS.**

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HALLIBURTON and Mott have noticed that choline has a characteristic physiological action when injected into the veins of animals, a marked lowering of blood-pressure being rapidly produced. This effect is either prevented or abolished, as the case may be, by the previous or subsequent injection of atropin.

Donath has asserted that a definite convulsive action is also produced upon the higher nerve centres, and has therefore concluded that it is an important factor in the production of the convulsions associated with epilepsy and general paralysis of the insane. He fails, however, to throw any light upon the question as to why there are no convulsions in other affections, such as disseminated sclerosis, diphtheritic paralysis, and tabes dorsalis, in which choline is also present in the blood. Donath is probably wrong in the supposition that there is any excess of choline in the blood in epilepsy ; if so, it can hardly be a factor in the production of the convulsions characteristic of that disease.

We have endeavoured to confirm Donath's experimental results, and also to learn the effect upon healthy animals of maintaining in their circulation for different periods various

amounts of choline, and have used rabbits and rats for our experiments.

The rabbits are denoted A, B, C, D, each weighing about 700 grams, and being about three months old at the beginning of the experiments. Of the rats A, B, C, A and C weighed 120 grams, and were about six weeks old; B weighed 260 grams, and was about six months old.

The rabbits B, C and the rat B were trephined and injected sub-durally, the rabbits A and D were injected intravenously, and the rats A and C intraperitoneally. The tables contain a record of the actual injections performed on the various animals.

We are greatly indebted to Dr J. W. H. Eyre, who made the injections for us.

*Rabbit A.*—Injected intravenously with 1 per cent. choline in 0·75 per cent. sodium chloride solution; later with 2 per cent. choline in a similar solution.

Day of Experiment.	Dose given.	Total Dose received.
1st Day	·3 c.c. 1 per cent. sol.	·003 gram.
3rd "	·4 "	·007 "
6th "	·5 "	·012 "
8th "	1·0 "	·022 "
10th "	1·0 "	·032 "
12th "	1·0 "	·042 "
14th "	1·0 "	·052 "
16th "	1·3 "	·065 "
28th "	1·0 c.c. 2 per cent. sol.	·085 "
23rd "	1·0 "	·105 "
26th "	1·0 "	·125 "
28th "	1·0 "	·145 "
30th "	1·0 "	·165 "
34th "	2·0 "	·205 "
38th "	1·5 "	·235 "
42nd "	2·0 "	·275 "
50th "	3·5 "	·345 "
54th "	3·5 "	·415 "
59th "	2·0 "	·455 "
62nd "	3·5 "	·525 "
66th "	3·5 "	·595 "
76th "	4·0 "	·675 "
80th " —Death.		

In the case of this animal no clinical effects were noticed until the 12th day, when diarrhoea appeared; this became rather more acute for about three weeks and persisted until the end.

About the 30th day, when the animal had received a total dose of .2 gram of the drug, it became dull and listless, the forelegs were bowed outwards, while the hind legs were kept widely separated on standing up. The effect of the injection on the 50th day of the large dose of .07 gram was to produce profuse salivation almost immediately. By this time the changes first noticed about the 30th day were much more pronounced, so that the animal could no longer stand upright, but assumed a peculiar crouching attitude. On skiagraphing the animal the wrist joints of the forelegs were seen to be almost completely dislocated. After this, the first large dose, the animal was very tottery for some time, but later doses had less effect. On the 75th day the general condition of the animal was distinctly bad; diarrhoea and marked exophthalmos were present; the forelimbs seemed to have lost almost all power, the chest nearly touched the ground.

The injection of .08 gram on the 76th day produced marked salivation, and within five seconds slight convulsions came on and persisted for about fifteen seconds; next day it seemed fairly well, but four days later it died, *i.e.* on the 80th day of the experiment.

Post-mortem nothing abnormal was to be seen in any of the organs, except some slight hypertrophy and dilatation of the heart.

A microscopic examination of the various viscera and central nervous system was made.

The condition of the joints was such as is not uncommonly seen in rabbits kept in confinement and cannot be attributed to the choline.

*Liver.*—Hepatic cells healthy on the whole, although they presented some slight cloudiness. The vessels were generally injected and there was some cellular exudation around the interlobular veins and bile capillaries.

*Kidneys.*—Slight cloudy swelling of cells of convoluted tubules. In other respects normal.

*Stomach and Intestines.*—Healthy.

*Heart Muscle.*—Showed no change.

*Lungs.*—Some scattered foci of broncho-pneumonia.



## NERVOUS SYSTEM.

*Nissl Method.*—A large number of sections from the cerebral cortex, from the mesencephalon, and from various levels of the spinal cord were examined by this method. The cells in all these regions showed absolutely no changes from the normal. There was no evidence of any pathological condition in the meninges, ependyma, or vessels, which were also examined in sections stained with hæmatomylin and eosin.

*Weigert-Pal Method.*—Sections of the cerebral cortex, mesencephalon, pons, medulla, and cord were normal in every respect.

*Busch Method.*—Sections from the cortex, mesencephalon, and medulla showed no changes. Sections from the thoracic cord presented diffuse but slight degeneration in the lateral, and to a less extent in the posterior columns. This was much less marked in the cervical region and was absent in the post-thoracic segments.

*Summary.*—The slight changes present in some of the viscera and in the white matter of the thoracic cord were probably due in part to malnutrition associated with the rachitic condition, and in part to the effect of choline in the process of excretion.

*Rabbit B.*—Sub-dural injection with 1 per cent. and later with 2 per cent. choline solutions.

Day of Experiment.	Dose given.	Total dose received.
1st day	0·2 c.c. 1 per cent. sol.	·002 gram.
6th "	0·5 "	·007 "
9th "	0·5 "	·012 "
12th "	·8 "	·02 "
14th "	·5 c.c. 2 per cent. sol.	·03 "
16th "	·5 "	·04 "
19th "	1·0 "	·06 "
24th "	1·0 "	·08 "
26th "	1·0 "	·10 "
28th "	·7 "	·114 "
30th "	·7 "	·128 "

On the 30th day the animal jerked its head violently at the moment of injection, with the result that the needle was driven back towards the cerebellum, its track being clearly seen after death. The animal was at once violently convulsed, nystagmus

was very marked, violent circus movements were performed, and there was great retraction of the head. Four hours later, as the condition had not improved, chloroform was given and the animal killed. Post-mortem the appearance of the viscera was normal, but a microscopic examination was also made. The convulsions were probably due in the main to the action of the choline, possibly also in part to the stimulation by the needle.

*Clinical Symptoms in B.*—As in the case of A, diarrhoea appeared about the 10th day. On the 12th day the animal jerked its head, and the needle entered the brain substance in the leg area, as shown by slight paralysis of the left leg, which cleared up completely ten days later. During this time intravenous injections were done instead of sub-dural ones, which were then resumed. After the injection on the 26th day the animal was very sluggish in its movements, and almost comatose for a short time (due to heart failure); it sweated profusely, and salivation was marked.

No fresh symptoms developed up to the time of the final injection.

#### HISTOLOGICAL EXAMINATION.

*Nissl Method.*—Sections from cortex and spinal cord showed no abnormalities.

*Busch Method.*—The cortex, pons, mesencephalon, medulla, and cord were examined, but the thoracic region of the latter was the only part to show any change. As in rabbit A, there was diffuse degeneration in the antero-lateral and posterior columns. This was more intense than in the previous animal, but never so marked as is seen resulting from gross experimental lesions; in other words, it had the appearance of being due to a slight toxic influence, and not that which is associated with Wallerian degeneration.

The adrenals, the spleen, and the kidneys were also examined, but with the exception of some slight cloudy swelling in the latter were not noticeably altered.

*Rabbit C* was injected sub-durally at the same times as Rabbit B, with equal volumes of normal saline solution as a control, but never presented any symptoms.

*Rabbit D.*—In the case of rabbits A and B, small doses were employed at first and slowly increased, the experiments extending over a considerable period of time (30 and 80 days). The effect of an initial large dose was now tried.

3.5 c.c. of a 2 per cent. solution were injected into a vein. No convulsions were produced, but the animal was profoundly affected. Salivation was very marked, the animal trembled and became very tottery, and sweated profusely; in the course of an hour it had completely recovered.

Three days later a like dose was again given, without, however, producing similar symptoms.

Six days after the second injection the animal died, after having had severe diarrhoea for three days.

As we did not obtain the body until two days after death, no post-mortem was performed.

*Rat A* (a control rat of the same brood was kept).—As the dose employed in the case of the rabbits had produced such slight symptoms, and the drug evidently was not highly toxic, relatively enormous doses were employed in the rats in order to see if any further symptoms could be obtained. The injections were made into the peritoneal cavity.

Day of Experiment.	Dose given.	Total Dose received.
1st day	1.0 c.c. 2 per cent. sol.	.020 gram.
5th "	1.3 "	.046 "
8th "	1.5 "	.076 "
11th "	1.5 "	.106 "
15th "	1.5 "	.136 "
18th "	1.5 "	.166 "
22nd "	1.5 "	.196 "
33rd "	1.5 "	.226 "
36th "	1.5 "	.256 "
40th "	1.5 "	.286 "
44th "	1.5 "	.316 "
48th "	1.0 c.c. 4 per cent. sol.	.356 "
52nd "	1.0 "	.396 "
55th "	1.25 "	.446 "
58th "	1.25 "	.496 "
61st "	1.25 "	.546 "
64th "	1.25 "	.596 "
69th "	1.0 c.c. 10 per cent. sol.	.696 "
Death.		

*Clinical Symptoms.*—About the 8th day diarrhoea appeared, but no other effect was produced until the large dose on the 69th day was given, when the heart beat almost instantly became

irregular and very feeble, and in two or three minutes assumed a Cheyne-Stokes character. Salivation and lachrymation were marked. In five minutes the animal became convulsed, and expired two or three minutes later.

*Post-mortem.*—All the viscera and peritoneum seemed quite healthy. On microscopical examination the viscera and central nervous system presented the following appearances.

*Nissl Method.*—Sections from the cerebral cortex, the mesencephalon, and spinal cord presented no evidence of any morbid change.

*Busch Method.*—Sections from the cortex, mesencephalon, medulla, various levels of the spinal cord, sciatic nerve, and muscles of the hind limb were examined and found to contain no degeneration.

*Hæmatomylin and Eosin.*—Sections from the medulla and spinal cord showed no change in the nervous tissue, in the meninges, or in the vessels. The heart muscle, skeletal muscle, liver, and kidneys were apparently healthy.

*Rat B.*—Injected subdurally and intraperitoneally.

Day of Experiment.	Dose given.			Total Dose received.
1st day	·25	c.c.	2 per cent. sol.	·005 gram.
4th „	·25	„	4 „	·015 „
7th „	·25	„	4 „	·025 „
11th „	·25	„	4 „	·035 „
15th „	·25	„	4 „	·045 „
18th „	·25	„	4 „	·055 „
28th „	·25	„	2 „	·060 „
31st „	·25	„	2 „	·065 „
35th „	2	„	2 „ (intraperitoneal)	·105 „
39th „	1·5	„	2 „	·135 „
43rd „	1	„	4 „	·175 „
47th „	1	„	4 „	·215 „
50th „	1·25	„	4 „	·265 „
53rd „	1·25	„	4 „	·315 „
56th „	1·25	„	4 „	·365 „
59th „	1·25	„	4 „	·415 „
64th „	1	„	10 „	·515 „
68th „	·15	„	10 „ (subdural)	·530 „
71st „	1·25	„	4 „	·540 „

Day of Experiment.	Dose given.	Total Dose received.
73rd day	·25 c.c. 4 per cent. sol.	·550 gram.
77th „	·25 „ 10 „	·575 „
80th „	·20 „ 5 „	·585 „
97th „	—Rat killed.	

*Clinical Symptoms.*—8th day, diarrhoea persistent. 40th day, animal becoming lethargic and slow in movements.

After large dose on 64th day, salivation and lachrymation marked; no convulsions.

After strong sub-dural dose on 68th day, tetanic convulsions instantly supervened; salivation marked; half an hour later, animal had recovered.

71st day: slight circus movements; stupor, but no convulsions.

77th day: slight circus movements in ten seconds; tetanic spasms of extreme severity supervened in one minute; violent spasms and convulsions lasted one hour, animal having to be restrained. If released it rushed blindly round room, and jumped wildly into the air. Recovery. No subsequent symptoms up to time of death.

*Post-mortem.*—No gross changes seen.

*Nissl Method.*—Sections from the cortex, mesencephalon, pons, medulla, and spinal cord were examined and found normal.

*Weigert-Pal Method.*—Sections from the cortex, pons, medulla, and cord showed no change in the medullated fibres.

*Busch Method.*—No degeneration was found in sections from the cortex, mesencephalon, and cord.

*Hæmatorylin and Eosin.*—The heart, intestine, liver, and spleen were examined with negative results. The kidneys showed capillary engorgement and capillary hæmorrhages, with some cloudy swelling of the convoluted tubules.

In order to determine the approximate fatal dose for these animals, Rat C was given ·7 c.c. of a 10 per cent. solution of choline intraperitoneally. Salivation and lachrymation became very profuse, and the heart beat intermittent. In two or three minutes slight convulsions supervened, but in ten minutes the animal had practically recovered.

The following day 1 c.c. of a 10 per cent. solution was given. The resulting symptoms were similar but more severe, the animal recovering in about an hour. Four days later the same dose was repeated. Tonic convulsions were produced, and respiration failed

in five minutes, although the heart continued to beat for an hour. Post-mortem, no gross changes were detected.

#### CONCLUSIONS.

1. The repeated introduction of moderate doses of choline into the circulating fluids of an animal produces neither convulsions nor paralytic phenomena.

2. The introduction of very large doses of the same substance produces convulsions, but the doses necessary are relatively greatly in excess of what can be produced in the human subject by the ordinary degenerations of the central nervous system.

3. It is improbable, therefore, that the convulsions of general paralysis or of epilepsy are directly or solely produced by the presence of choline in the blood or cerebro-spinal fluid.

4. The presence of considerable quantities of choline in the circulation does not produce morbid changes of importance in the central or peripheral nervous system or in the visceral organs of the body.

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### **POSTERO-LATERAL DEGENERATION OF THE SPINAL CORD, OF VASCULAR RELATION, ASSOCIATED WITH SEVERE ANÆMIA.**

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and

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THE following case, of well-marked degeneration in the posterior and lateral columns of the spinal cord, presents several points of considerable interest. A prominent feature of the degeneration was its relation to the distribution of the spinal blood-vessels.

#### CLINICAL HISTORY. (By D. R.)

John T. H., aged 49, roller coverer, came under the treatment of one of us (D. R.) on account of anæmia, pain in the back, and weakness in the legs.

His health had been good until twelve months before he came under treatment. About that time he commenced to suffer from pain in the back, and a feeling of "electric shocks" running down into the tips of the fingers and toes whilst walking. There

was, however, no discomfort when the patient was sitting still at his work. These symptoms continued, and soon he began to suffer also from a sensation of tingling in the fingers. This caused him to have great difficulty in attending to his work.

For three weeks before he came under treatment he had suffered from girdle pains round the abdomen, and from a sensation of constriction round the thighs. The latter symptom was the better marked, and continued until the termination of the disease.

The left leg had been paralysed from childhood, owing to infantile paralysis (*anterior poliomyelitis*).

When *first examined* (February 1903), the patient was very anæmic. The lungs were normal; the heart sounds were feeble, but no murmur was present. The urine was normal.

The tongue was furred, there was a sensation of sickness after food, but no vomiting occurred. The bowels were constipated.

There was no Argyll-Robertson pupil. The pupils reacted normally.

There was great pain and tenderness over the lumbar region of the spine, but none in the dorsal or cervical region.

[There were the signs and symptoms of an old infantile paralysis (*anterior poliomyelitis*) of the left leg, the muscles below the knee having been wasted and paralysed since infancy.] The knee-jerks at this date (February 1903) were both absent. There was no ankle-clonus. The superficial reflexes were present.

Mercury and iodide of potassium were given, and a blister applied over the painful region of the spine; but no improvement occurred, and spastic paraplegia gradually developed. The legs became rigid, the reflexes increased, and walking became impossible. On May 24 the knee-jerks were present on both sides. Well-marked ankle-clonus was obtained on the right side. On the left side (side of old infantile paralysis) it was not obtained. The plantar reflex on the right side was of the extensor type (Babinski's reflex). There was hyperæsthesia of the legs and abdomen, and a feeling of tingling in the fingers. By this date the pain in the back had disappeared.

The weakness and anæmia steadily increased. On April 16 the amount of hæmoglobin was 50 per cent. of the normal,

and the red blood corpuscles were 1,860,000 per cubic millimetre. On May 24 the hæmoglobin was 22 per cent. and the red blood corpuscles 1,120,000 per cubic millimetre. There was no excess of leucocytes.

During the summer of 1903, the condition remained much the same; but the ankle-clonus became more marked and the knee-jerks much exaggerated. Hyperæsthesia and pains in the legs became so troublesome that the bed-clothes had to be raised from the limbs by a cradle. Frequent and severe vomiting occurred in the first two weeks of July, and constipation became very troublesome. But there was no anæsthesia and no affection of the bladder up to the fatal termination of the disease, which occurred on July 15.

(The spinal cord was removed post-mortem by D. R.)

Clinically the symptoms were those of spastic paraplegia of gradual onset, with hyperæsthesia of the legs, pain in the back and legs, and severe anæmia.

#### MICROSCOPICAL EXAMINATION. (By R. T. W.)

The spinal cord was hardened in Müller's fluid. On transverse section, after hardening, marked degeneration could be detected even by the naked eye in the posterior and lateral columns, the degenerated parts being paler in colour than the rest of the white matter. These pale areas corresponded to the degenerated parts recognised on microscopical examination (see figures).

Pieces of cord were embedded in celloidin. Sections were stained according to the methods of Weigert-Pal, van Gieson, Marchi, Bielschowsky, and Mallory, and also with aniline blue-black and logwood and eosin. Tracts and patches of degeneration were seen in the lateral pyramidal tracts and in the posterior columns, from the upper cervical to the sacral region. In both lateral and posterior columns the changes were most marked in the cervical region and least in the sacral (see figures).

In the sacral and lower lumbar regions the usual changes associated with an old infantile paralysis could be seen in the left anterior horn of grey matter. [The left leg had been paralysed since infancy, and the symptoms and history indicated an acute anterior poliomyelitis at that period.] The left anterior horn



of grey matter was slightly smaller than the right. At its outer part was a semi-circular patch, which appeared, even to the naked eye, paler than the rest of the grey matter in the Weigert's sections (see Fig. 4). Under the microscope, in this pale area, the fine nerve fibres of the grey matter were greatly diminished in number, whilst in the adjacent part of the grey matter the fine nerve fibres were very numerous. In this area, in which the fine nerve fibres were almost absent, the nerve cells had also disappeared; and the neuroglia connective tissue was increased in amount, stained more deeply, and contained more nuclei.

*Distribution of Degenerative Changes in the White Matter.*—The changes in the lateral columns (seen as pale areas in the Weigert's specimens) were in the region of the crossed pyramidal tracts. This lateral degenerated area was separated from the surface of the cord in the cervical and upper dorsal regions by a narrow zone of normal or only slightly degenerated tissue. The degeneration was not always strictly limited to the crossed pyramidal tract in the cervical and dorsal regions. In some section in the dorsal region there was a separate small round patch in the white matter just in front of the crossed pyramidal tract (Fig. 2). Also in many of the sections of the dorsal and cervical cord, the degenerated area in the lateral columns had not exactly the shape and size of the crossed pyramidal tract, and sometimes did not affect the whole of the tract; but the degeneration often appeared as round patches or streaks surrounding the blood-vessels. In the lumbar and sacral regions the lateral degeneration was localised to the crossed pyramidal tracts, and had the size and shape of these tracts (Figs. 1 to 4).

In the posterior columns of the cervical region there was marked degeneration of Goll's columns, with irregular patches of degeneration in Burdach's columns. The most posterior part of Goll's columns, adjacent to the surface of the cord, was less degenerated than other parts of these columns. The white matter adjacent to the posterior grey horns and commissure (*i.e.* the posterior root zones and the posterior ventral fields) was not degenerated. Evidently the changes in Goll's columns in the cervical region were chiefly secondary to changes in the posterior columns of the cord at a lower level.

In the lower dorsal region the degeneration in the posterior



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 7.

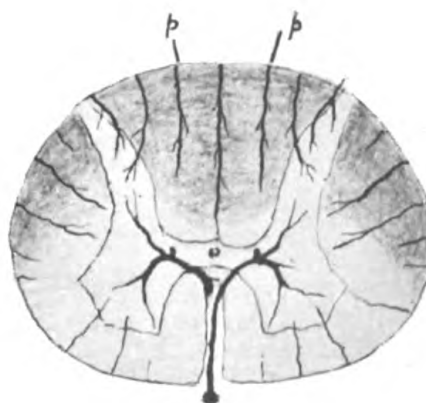


FIG. 8.



columns was localised around the course of the posterior intermediate septal artery, which appeared to be surrounded by a sheath of degenerated white matter (see Figs. 3 and 6). At some parts there were also irregular or more or less circular patches in Burdach's columns. Goll's columns, in the lower dorsal region, were unaffected at the posterior third. Burdach's columns were also unaffected near the surface of the cord, and in the parts adjacent to the posterior grey matter and the posterior commissure.

In the lumbar and sacral regions the degeneration diminished in the posterior columns, but it continued to be localised chiefly around the posterior intermediate septal artery. In the sacral region this degeneration in the posterior columns was very slight: it was better marked on the right side than on the left. In the posterior columns of the sacral, lumbar, and lowest dorsal regions there was an area of normal fibres between these degenerated patches and the posterior median fissure. In the sacral region the changes in the lateral columns were very slight, and were localised to the crossed pyramidal tracts. In all regions of the cord the following parts were unaffected by degeneration and appeared normal:—The meninges, the intramedullary fibres of the posterior and anterior nerve roots, the posterior root zone, Lissauer's zone, the anterior and antero-lateral white matter, the anterior and posterior horns, Clarke's columns and other parts of the grey matter (except the left anterior horn in the lumbo-sacral region, which, as already mentioned, presented the changes that are associated with an old anterior poliomyelitis).

#### APPEARANCE OF DEGENERATED AREAS.

Three forms of degenerative changes were seen. (1) In one form, in the degenerated area, nerve fibres had for the most part entirely disappeared, leaving empty spaces in the neuroglia. These spaces were often very large, much larger than the area of the transverse section of several (10 to 20) nerve fibres. The neuroglia in this form of degenerated patches was not increased, but appeared to be diminished in amount. The few nerve fibres which remained at the periphery of these patches often presented enormously dilated myelin sheaths, and sometimes also swollen axis-cylinders. This was the appearance of the degenerated areas in the lateral

columns at most parts of the cervical and dorsal regions, and also of the degeneration in the posterior columns in the lumbo-sacral and lower dorsal regions, and at many parts of the upper dorsal and cervical regions (see Fig. 5).

(2) In other patches in the degenerated areas the nerve fibres had disappeared. Spaces were left in the neuroglia at the parts at which nerve fibres had been previously situated, but in addition there was a great increase of the neuroglia—an actual sclerosis. This appearance was seen in Goll's columns in the cervical region, and at some parts of the lateral and posterior columns in the dorsal region.

[In both of these forms of degenerated areas Marchi's method of staining revealed only few degenerated nerve fibres (stained black) at the periphery of the patches. In the more central portions of the patches probably all products of degenerated fibres had become absorbed.]

(3) In a third form in the degenerated areas there was only a slight increase of neuroglia, with a few empty spaces, from which nerve fibres had disappeared: but Marchi's stain showed numerous black-stained products (black dots) of degeneration of the nerve fibres. These areas were probably at a less advanced stage of degeneration. The crossed pyramidal tracts of the lumbar and sacral regions presented this appearance.

In all of the forms of degenerated areas the diseased patches appeared pale in sections stained according to Weigert or Pal's method, owing to the absence of normal black-stained myelin sheaths of the nerve fibres. Sections stained with formalin and silver nitrate (Bielschowsky's method) showed that the axis-cylinders had also disappeared in the diseased areas, except just at the periphery, where sometimes swollen axis-cylinders were seen. (In this respect, therefore, the patches differed from those of disseminated sclerosis, in which numerous naked axis-cylinders are seen, when sections are stained according to Bielschowsky's method.) The degeneration in the crossed pyramidal tracts of the lumbo-sacral region, and in Goll's columns of the cervical region, presented the appearance and localisation of secondary degeneration (such as are seen after any transverse lesion of the cord). But in other regions—in the lateral columns of the cervical and dorsal regions, in the posterior columns of the dorsal and lumbo-sacral region, and in Burdach's columns of

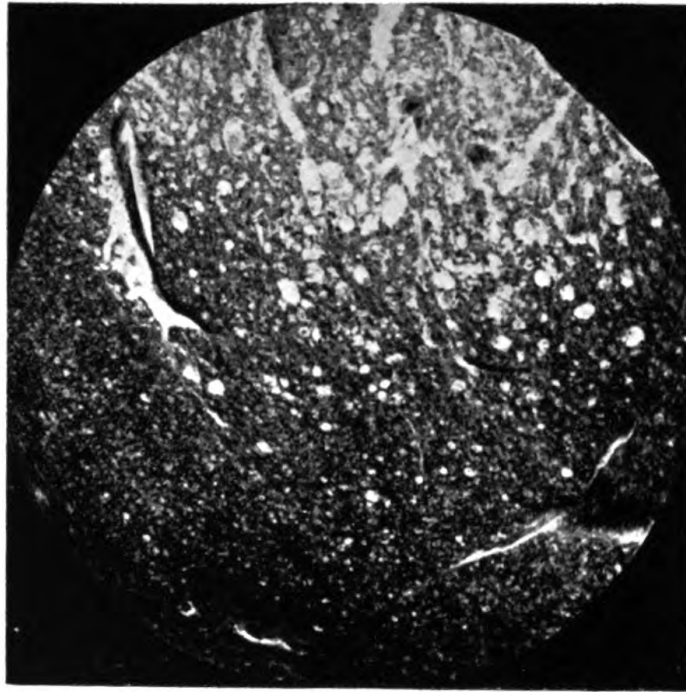


FIG. 5.



FIG. 6.



the cervical region—the degeneration was somewhat irregular, and was not strictly limited to any system of fibres. Numerous dilated blood-vessels were seen in these degenerated areas last named. The external coats of the vessels were often greatly thickened, and their perivascular sheaths were often greatly dilated. (These changes were well seen in sections stained according to Van Gieson's method, see Fig. 7.) The nuclei of the vessel walls were increased in number.

#### RELATION OF CHANGES TO BLOOD-VESSELS.

The degeneration in the crossed pyramidal tracts of the lumbo-sacral region and in Goll's columns of the cervical regions appeared to be of the nature of secondary degeneration, as already mentioned. But in other parts, *i.e.* in the posterior columns of the dorsal and lumbo-sacral regions, and in the lateral columns of the cervical and dorsal regions, the degeneration was clearly related to the course of blood-vessels. Often there were, in these columns, round patches of degeneration, with one or more greatly dilated vessels in the centre; or streaks of degeneration followed the course of a dilated vessel (see Fig. 6). In the posterior columns of the lower dorsal and lumbo-sacral regions the degeneration was clearly localised around the course of the posterior intermediate septal arteries (see Figs. 3, 6, and 8). The transverse section of the normal spinal cord may be divided into two areas: one supplied by the posterior arteries, the other by the anterior arteries (see Fig. 8). In the case here recorded the changes were clearly localised to the region supplied by the posterior arterial system of spinal arteries, *i.e.* to the shaded area in Fig. 8.

#### SUMMARY.

Clinically the case was one of spastic paraplegia, with slight sensory symptoms, associated with severe anæmia. Pathologically the spinal changes consisted of degeneration of the white matter in the posterior and lateral columns of the spinal cord, *i.e.* in the region supplied by the posterior arterial system. The degeneration was clearly related to the distribution of the blood-vessels of these areas, and was not localised to any neurone system. A peculiar feature of the degeneration, in many of the patches, was



its vacuolated appearance—very large spaces being present from which nerve fibres had entirely disappeared, whilst the neuroglia connective tissue had diminished in amount.

In recent years many cases of combined postero-lateral spinal degeneration have been recorded, in which there was marked anæmia, or the symptoms of pernicious anæmia during life. It appears probable that both the anæmia and the spinal degeneration are caused by some toxic substance present in the blood, but the nature of this toxin is not yet known.

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(In these articles references are given to most of the previous papers on the subject.)

#### DESCRIPTION OF ILLUSTRATIONS.

Fig. 1.—Microphotograph ( $\times 4$  diameters) of cervical region of cord. Weigert-Pal's stain. Pale areas = degeneration.

Figs. 2 and 3.—Dorsal regions.

Fig. 4.—Lumbar region (same stain).

Fig. 5.—Microphotograph of white matter of lateral column. At upper part (paler portion) there is marked degeneration of nerve fibres, and large spaces are left from which the nerve fibres have disappeared, causing a vacuolated appearance (Weigert-Pal's stain).

Fig. 6.—Microphotograph of posterior columns, showing streak of degeneration (pale) in the course of the posterior intermediate septal artery (this degenerated pale area runs obliquely through the centre of the photograph). To the right of the degeneration is the posterior median fissure.

Fig. 7.—Drawing showing great thickening of the walls of small blood-vessels, and marked dilatation of the perivascular sheath, at a degenerated region in the white matter (van Gieson's stain).

Fig. 8.—Diagram showing distribution of anterior and posterior arterial system of the spinal cord. Area of white matter supplied by posterior arteries is shaded. P=posterior intermediate septal artery.

# Abstracts

## PHYSIOLOGY.

**INVESTIGATIONS IN THE MOTOR LOCALISATION OF THE**  
**(210) INFERIOR EXTREMITY IN THE HUMAN CORD.** (Unter-  
 suchungen über die motorische Localisation der unteren Ex-  
 tremität im Rückenmark des Menschen.) C. PARHON und  
 M. GOLDSTEIN, *Neurol. Centralb.*, 1905, S. 498.

THOUGH this paper contains the results of new work, the facts it presents are chiefly arrived at by a compilation of the conclusions of earlier work of their own and of other authors.

The highest nerve supply of the muscles of the lower extremities is from the 3 L segment, and the lowest fibres come from the 3 S segment. As a rule each muscle is represented centrally by a single group or column of cells in the ventral horns. The following localisations seem to be fairly definite:—

*Quadriceps*—External group, 3 L and 4 L.

*Sartorius*—Antero-external group, 3 L and 4 L.

*Gracilis*—Central group, 3 L.

*Adductores brevis et longus*—Central group, 3 L.

*Adductor magnus*—Central group, 4 L.

*Semi-membranosus*—Central group, inferior, 4 L and 5 L.

*Semi-tendinosus*—Postero-internal central group, 1 S.

*Biceps cruralis*—Antero-external central group, 1 S.

*Tibialis anticus*—Posterior group, inferior, 4 L and 5 L.

This is the leg muscle, which is the highest represented in the cord.

*Gastrocnemius*—Post-central group, 5 L to 2 S.

*Flexors of Toes*—Posterior group, 5 L.

*Peronei*—External group, 1 S.

*Plantar muscles*—Post-postero-lateral group, inferior, 1 S and 2 S.

GORDON HOLMES.

**THE EFFECT OF COLD NARCOSIS ON REFLEX ACTION IN**  
**(211) WARM-BLOODED ANIMALS.** SUTHERLAND SIMPSON and  
 PERCY T. HERRING, *Journ. of Physiol.*, 1905, p. 305.

DURING deep anæsthesia, the heat-regulating mechanism is interfered with, and a warm-blooded animal tends to take the temperature of its surroundings. In these experiments cats were used.

They were fully anaesthetised with ether, and placed in a chamber the temperature of which was maintained at about 4° C. by being surrounded with an ice-jacket. The temperature, as indicated by a thermometer in the rectum, gradually fell and when it had reached 23° C. to 24° C. the animal was in a condition of cold narcosis, and the administration of ether was no longer necessary. At 16° C. the respiratory movements and heart-beats were almost imperceptible, no reflexes could be elicited, and at first sight it was difficult to determine whether the animal was alive or dead.

A very noticeable feature in cats which had their temperature reduced in this way was the increase of irritability of the skeletal muscles to direct stimulation. Tapping the muscles brought about an immediate and well-marked contraction; this increased as the temperature was lowered, and it did not disappear at the death of the animal, being sometimes very evident two and a half hours after death.

The knee-jerk was a very persistent phenomenon. It was, as a rule, easily elicited, and it was present after all the reflexes had disappeared. The lower the temperature the smaller the jerk; with rectal temperatures from 15° C. to 19° C. it was feeble and sluggish, but quite evident. It disappeared simultaneously with the death of the animal.

To facilitate the observation of the reflexes and to hasten the recovery of the animal from cold narcosis, it was removed from the cold chamber when its temperature had been sufficiently reduced and placed in a warm bath. The temperature soon began to rise, and recovery was comparatively rapid. The reflexes were tested at frequent intervals, and the rectal temperature at which each one appeared was noted.

During recovery from cold narcosis, one of the first reflexes to reappear was winking of the eyelids when the skin at the inner angle of the eye was gently stroked; this occurred at an average temperature of 22° C.

The application of hot water to the skin of the paws was found to be a very efficient stimulus; its first effect was to accelerate respiration, and it did this when the rectal temperature was about 22° C. No attempt was made to withdraw the limb till the temperature was above 24° C.

Pinching the skin did not provoke any response till the temperature was 25° C.

The pupil did not begin to contract to light till the temperature had reached 24° C., and then only very slowly.

Shivering and rhythmical swimming-like movements of the limbs began at a rather variable temperature, in some animals below 22° C., in others not till 27° C.

A reflex movement of the ear when the hairs inside it are

touched, usually found in cats, returned at a rectal temperature of about 24° C.

The conjunctival reflex was the last to appear, and that at a fairly constant point between 26° C. and 27° C.

Voluntary movements and consciousness appeared shortly after the conjunctival reflex was obtainable, but the power of co-ordinating movements was not regained till the temperature had risen above 30° C.

These results were compared with similar ones obtained from cats during recovery from deep ether anæsthesia, and the order of reappearance of the reflexes in the two conditions agrees very closely. The knee-jerk is abolished when the ether anæsthesia is deep, but rapidly returns. The application of heat to the skin appears to be the most efficient stimulus, and first causes respiratory acceleration. The conjunctival reflex is the last to reappear.

The anæsthesia induced by reducing the rectal temperature to 23° C. or lower is just as effective as that produced by ether or chloroform, but the application of this method is too tedious to admit of its becoming one of practical importance.

AUTHOR'S ABSTRACT.

### **PATHOLOGY.**

**SOME ALTERATIONS OF THE CEREBRAL TISSUE DUE TO THE**  
(212) **PRESENCE OF TUMOURS.** (De quelques altérations du tissu cérébral dues à la présence de Tumeurs.) R. WEBER et A. PAPADAKI, *Nouv. Icon. de la Salpêtrière*, mars-avril 1905, p. 140.

THIS investigation is founded on the examination of the brains of five cases of intracranial tumour. The growths were situated in different regions, in the occipital and frontal lobes, in the middle cranial fossa, in the mid-brain and pons, and in the ponto-cerebellar angle, yet the direct and indirect pathological effects were more or less similar, though in part influenced by the site of the lesion. They were: ventricular dilatation, always greatest in the anterior horn of the lateral ventricle; lymph stasis; dilatation of the perivascular spaces, and occasionally exudate and hæmorrhage into these; and finally, rarefaction and vacuolation of the white substances, chiefly in the short association tracts.

The conclusions the authors venture to draw are:—

(1) The lymph current of the brain flows from the periphery towards the ventricles, and thence through the aqueduct of Sylvius to the spinal sub-dural space, and the general effect of a tumour varies as it interrupts this or not. To interruption of the lymph flow the early fatality of sub-tentorial tumours is to be attributed.

(2) Increased intracranial pressure is not equally distributed.

It is greatest in the neighbourhood of the tumour, next around the ventricles if these be dilated, and it gradually diminishes away from the tumour owing to the resistance the cerebral tissue exerts to its propagation.

(3) Under the influence of excessive intracranial pressure the convolutions first close up the fissures, then they sclerose, and finally their fibres atrophy. These changes are greater in the white than in the grey matter. This is the reverse of the pathological changes in general paralysis, and clinically, too, the dementia associated with the late stages of intracranial neoplasms is not identical, or even similar, to that of general paralysis.

(4) The short association fibres suffer more severely in intracranial pressure than the projection tracts.

(5) When a unilateral tumour obstructs the ventricular circulation, it is as a rule the opposite ventricle which dilates the more.

(6) Dilatation of, and exudation into, the perivascular spaces appears as a rule in cases of tumour, but it never results in inflammatory change or necrosis.

(7) The lymph stasis does not depend on interference with blood circulation. There is probably a special nutritional system in the brain not yet recognised. The assumption of auto-intoxication resulting from tumours is baseless. On the other hand, auto-intoxication may result from the imperfect removal of effete products due to the lymph stasis.

GORDON HOLMES.

#### ON SOME CHARACTERS OF SPECIFIC ANTITOXINS IN THE

(213) **BLOOD-SERUM OF EPILEPTICS.** (Ueber einige Charaktere spezifischer Antitoxine im Blutserum der Epileptiker.) CARLO CENI, *Centralb. f. Nervenheilk. u. Psychiatrie*, Bd. xxviii., 1905, S. 372.

THE author has previously demonstrated that the epileptogenic poison, having no special toxic action on other animal species, is specific for man, and that the toxicity of the serum is proportionate to the severity of the disease. In the status epilepticus, more especially, the serum is hypertoxic and may be as potent as the specific antiserum.

He now records investigations on the degree of constancy of natural antitoxic substances in the serum of epileptics, in relation to the severity of the disease, and draws the following conclusions:—

1. The antitoxic action of the serum of epileptics against the specific antiserum is not materially modified during the various phases of the disease in its natural course.

2. In severe cases of epilepsy, and especially during periods in

which the disease is aggravated (status epilepticus, etc.), the anti-toxin of the serum is markedly reduced and may vanish therefrom.

3. If an epileptic be repeatedly injected with antiserum or hypertoxic serum of epileptics, his morbid condition is aggravated, and simultaneously his blood-serum manifests diminished antitoxic power against the specific antiserum. W. T. RITCHIE.

### CLINICAL NEUROLOGY.

**FRIEDREICH'S DISEASE AND HEREDITARY CEREBELLAR**  
(214) **ATAXIA.** (*Maladie de Friedreich et Hérédoataxia Cérébelleuse.*) F. RAYMOND, *Nouv. Icon. de la Salpêtrière*, jan., fév. 1905, p. 5.

THE object of this paper is to prove that it is unnecessary and misleading to multiply special types of familial ataxy, and, by demonstrating the occurrence of intermediate varieties, to show that the most widely distinct forms can be linked to each other by an almost continuous series of connecting cases.

Beginning with the description of a case which is, in most respects, typical of an ordinary Friedreich's ataxia, the author recounts the various lesions that have been found in similar instances, showing that the morbid process may not be confined to the posterior columns of the cord, but may implicate other portions also. These various deviations from the typical lesion are paralleled by numerous clinical instances where some of the ordinary symptoms are absent, and where unusual ones are present.

The view promulgated by Senator regarding the involvement of the cerebellum is next discussed, and in this connection reference is made to the distinct type known as Cerebellar Hereditary Ataxia which was described by P. Marie.

The most valuable part of the paper, however, is the concluding section, which contains a full account of a case, intermediate between ordinary Friedreich's ataxia and Marie's cerebellar ataxia, that had been under prolonged observation in Prof. Raymond's clinique. During life this patient exhibited symptoms characteristic of ordinary Friedreich's disease, but the post-mortem examination, which was conducted with great care by Philippe and Oberthür, disclosed changes in the cerebellum as well. These consisted in a degenerative atrophy of the cellular elements. The degeneration was in active progress at the time of the patient's death, and was not associated with any distinct sclerosis. The nerve fibres related to the affected cells were notably reduced in number, the defect being continued through the cerebellar peduncles and extending to the spinal cord. The cord itself showed changes not only in the posterior columns, but also in the antero-lateral tracts

and in the grey matter of the anterior cornua. The posterior roots were involved, but, apart from thickening of their fibrous sheaths, the posterior ganglia were only slightly affected, and the peripheral nerves were found free from any marked degeneration. Changes were noted in the meninges and vessels of the cord.

This case, therefore, whilst clinically resembling a pure Friedreich's ataxia, must be pathologically classed with those recorded by Menzel, Meyer, and others.

The author's conclusions may be summed up as follows:—Friedreich's disease, Marie's cerebellar ataxy, and the intermediate forms constitute morbid types having as a point in common the implication of the cerebellar system in its centres, or in its afferent and efferent tracts, and the term "Cerebellar Ataxia" should be employed in a wider sense to indicate the common syndrome—cerebellar gait, static ataxia, inco-ordination, and nystagmus—which combines in one comprehensive group the separate types of the disease. One may then enumerate the following subdivisions:—

- (1) A *spinal* type (ordinary Friedreich), with absence of reflexes, scoliosis, and club-foot.
- (2) A *cerebellar* type, with optic atrophy, vertigo, and mental implication, added to the common syndrome.
- (3) A *medullary* type, with vomiting, dyspnoea, and cardiac arrhythmia.
- (4) A *ponto-medullary* type, with auditory troubles.
- (5) A *generalised* type.

In support of this grouping it is pointed out that many other nervous diseases show variations due to different localisations of the lesion. Thus, in addition to ordinary locomotor ataxia, there are cases where implication of the medulla and encephalon materially affect the general character and symptoms of the disease. Or, again, there are several aberrant types of insular sclerosis; and yet again, in muscular atrophies, one finds the myopathic and myelopathic types brought into connection through the form known as the Werdnig-Hoffman type.

The author is disposed to carry his reasoning a step further. As familial ataxies vary according to the localisation of the lesion in the cerebellar system, one may trace further variation by the extension of the hereditary lesion to other systems of the brain and cord, passing by intermediate cases from cerebellar ataxias to certain paraplegias on the one hand, and to myelopathic muscular atrophies on the other, whilst an upward extension of the lesion to the higher centres may correspond to mental defect and to a hypoplasia, without sclerosis, of the whole cerebro-spinal nervous system.

HARRY RAINY

**TABES UND SYNERGIE.** G. CATÒLA und M. LEWANDOWSKY  
(215) *Centralb. f. Nervenheilk. u. Psych.*, May 1905, p. 369.

IN contradistinction to Foerster, the authors find in making tabetics close the fist with the hand in a drooping position, there is no loss of co-ordination between the wrist extensors and finger flexors. This in cases even with marked disturbance of the muscular and stereognostic sense and complete anæsthesia. Synergy is only complete when the movement is an impulsive one, not when an attempt is made to carry out certain defined movements. Co-ordination is therefore not dependent on centripetal sensory impulses, but as Leyden, Goldschieder, and others hold, is predetermined centrally. J. H. HARVEY PIRIE.

**CONCERNING A VIEW OF THE LESS KNOWN TYPES OF**  
(216) **DISSEMINATED SCLEROSIS.** (Ueber einige weniger bekannte Verlaufsformen der multiplen Sklerose.) EDUARD MÜLLER, *Neurolog. Centralbl.*, July 1, 1905, p. 593.

EDUARD MÜLLER has recently published a valuable monograph on multiple sclerosis, embodying the large experience of the Erlangen Clinique. In the present paper he briefly describes some types of the disease which are apt to escape recognition.

Charcot and his school may be said to have painted the classical picture of disseminated sclerosis; during recent years, however, our knowledge of the symptomatology has been greatly extended, and those cases which conform to the description of the disease as it was known twenty-five years ago now form a comparatively small proportion of the whole as at present diagnosed.

The author indicates that there are three important facts, the recognition of which has considerably modified our conception of the disease.

Firstly, we now know that the affection is a comparatively common one. Thus, according to Strümpell, it is in rural districts the most common gross organic affection of the brain and spinal cord, while in the large towns it ranks in frequency only second to the syphilitic and parasymphilitic affections.

Secondly, the "*formes frustes*" of the disease are far more common than the classical cases, and at the same time they present a typical symptom complex which permits of a diagnosis as certainly as do those cases in which the classical tripod is present.



Thirdly, pallor of the optic discs is a much more valuable sign, both on account of its frequency and special characters, in the diagnosis of disseminated sclerosis, than are the so-called classical symptoms of the disease. The author holds that the peculiar affection of the optic nerve (simple atrophic pallor of the papilla, particularly of the temporal half) is the most important sign of disseminated sclerosis, presenting itself, as most authorities will agree, in about fifty per cent. of the cases. The disparity between anatomical changes and disturbance of function, which is often such a striking feature in disseminated sclerosis, is well exemplified in the optic nerves.

The author agrees with Strümpell in attaching great weight to absence of the abdominal reflexes as a diagnostic sign of the disease, even in its earliest stages. He states that in young healthy persons, in whom there is no acute abdominal condition, no excessive development of fat, œdema, or abnormally lax condition of the abdominal walls, these reflexes are constantly to be obtained if examined for with due precautions. On examining the abdominal reflexes in 1000 soldiers on one occasion only, they were found to be present on both sides in every case with a single exception.

A class of case which is apt to escape detection is one which commences suddenly with amblyopia, it may be complete blindness, usually confined to one eye, and generally clearing up entirely. With this somewhat uncommon type, thanks to the writings of Dr Buzzard, English neurologists are well acquainted. The subjective symptoms may entirely disappear and some pallor of the optic discs, absence of the abdominal reflexes, a bilateral Babinski sign, and perhaps some slight unsteadiness of the hands in carrying out fine movements may alone remain as indications of the existence of the disease.

A comparatively common type is the case which begins with a localised feeling of tiredness, sometimes present, sometimes absent, which may exist for many years without further subjective sensations.

Very uncommonly cases are met with which are characterised by severe neuralgic pains, most usually associated with muscular spasms, sometimes occurring independently. These cases are very exceptional.

In spite of the numerous modes of onset, the physical signs in the great majority of cases present a very striking similarity. In difficult cases an accurate and careful history often affords at least as important information for diagnosis as does the physical examination.

EDWIN BRAMWELL.

**ON MENTAL DISORDERS, ESPECIALLY THOSE OF THE INTEL-**  
**(217) LECT, IN MULTIPLE SCLEROSIS.** (Ueber psychische,  
insbesondere Intelligenzstörungen bei multipler Sklerose.)  
W. SEIFFER (of Berlin), *Arch. f. Psych.*, Bd. 40, H. 1, p. 252.

IN the published cases of multiple sclerosis, mental disorders are frequently mentioned, but the current opinion is that these disorders are only slight, even in the last stages of the disease. The usual mental changes described are an enfeeblement of the intellect and a more or less marked euphoria. Numerous authors have described the mental condition of their patients as normal.

Seiffer subjected ten consecutive cases of multiple sclerosis to a systematic examination of their mental condition, and more especially of their intellect. In this examination he followed the methods of Ziehen's clinic, investigating in each patient the store of simple concrete ideas, and of abstract ideas, the power of attention, the reaction time, and the association of ideas; ten different tests were employed in studying the last.

In only one case could no defect be demonstrated. The other nine cases showed considerable disorder of the intellect; this disorder affected especially the concrete ideas derived from the remote and recent past, and the association of ideas. The power of attention and the grasp of abstract ideas were frequent, but not so constantly impaired.

The patient, who showed no disorder of the intellect, was extremely interesting, as she had previously had an attack of hallucinatory confusion (Amentia) with complete recovery.

Seiffer considers that this psychosis was probably due to a sclerotic brain process, and not a casual complication of the disease. He discusses the question of the connection between the duration of the disease and the defect of the intellect; in his cases the extent of the mental disorder did not run parallel with the duration of the disease.

The spinal forms of the disease present few mental symptoms in comparison with those found in the cerebral, cerebro-spinal, and bulbar forms.

The nature of the defect of the intellect which is especially seen in defective memory and association of ideas and in a certain slowness of thinking, is insufficient by itself to distinguish the dementia due to multiple sclerosis from that on an alcoholic, epileptic, or other basis; but it is characteristic when taken in association with the euphoria and labile mood of the patient.

The observations published in this communication gain much

by the clearness of their presentation ; the methods employed are definitely and concisely stated, the cases well summarised, and the final conclusions are clearly brought out.

C. MACFIE CAMPBELL.

**DIFFUSE SARCOMATOUS INFILTRATION OF THE SPINAL  
(218) PIA MATER.** STANLEY BARNES, *Brain*, 1905, p. 30.

THE author describes in detail two cases, and gives an account of nine others reported by different observers. He holds that "in general it remains impossible to give any accurate diagnostic signs of the condition ; but it seems clear that if a patient who is known to present the signs of unlocalisable cerebral tumour develops well-marked pains in the limbs and trunk, with early loss of deep reflexes and rapid wasting, the presence of disseminated growth in the spinal pia and posterior root ganglia should be suspected. In such cases it would be well to examine also the ventricular lining in case the less obvious gelatinous growths which occur in this situation may be overlooked."

Barnes comes to the following conclusions:—

1. If a growth originates in the caudate nucleus or in any other part of the brain, and eventually finds its way to the lining of the ventricular cavities, it may "infect" the cerebro-spinal fluid. Metastatic growths may then occur in the following situations:—

- (1) In the lining walls of the lateral, third or fourth ventricles ;
- (2) in the meninges (sub-arachnoid) at the base of the brain ;
- (3) in the pia mater and arachnoid around the spinal cord ; and
- (4) in the posterior root ganglia, particularly of the cauda equina.

2. In all probability diffuse sarcomatous infiltration of the spinal pia arachnoid, which is occasionally found post-mortem, is in all cases the expression of a sarcomatous infection of the cerebro-spinal fluid as a result of some primary growth which lies exposed to the stream of the cerebro-spinal fluid high up in the nervous system.

3. The regions occupied by such secondary growths are exactly the same as those inflamed by tubercular meningitis after the rupture of a tubercular "tumour" of the brain. EDWIN BRAMWELL.

**A CASE OF TRAUMATIC CERVICAL HÆMATOMYELIA AND  
(219) COMPLETE DIVISION OF THE CORD, WITH PROBABLE  
DISLOCATION OF THE FIFTH CERVICAL VERTEBRA.**

Remarks on the Location of the Centre for the Biceps Reflex.

C. S. POTTS, *Journ. of Nerv. and Ment. Dis.*, June 1905, p. 359.

A MAN, age 56, after a fall downstairs, was unconscious for about twelve hours. He had complete flaccid motor paralysis of legs

and trunk, and also of the arms, with the exception of shoulder movements, diminished power of flexion, and slight power of supination of forearms; no paralysis of cranial nerves. Sensory paralysis (touch, pain, temperature) was complete on legs, trunk, ulnar side of arms and forearms, and whole of hands except thumbs. All tendon reflexes (triceps, wrist, knee, and Achilles tendon reflexes) were absent except the biceps reflex, which remained very active on the left side till death, but was lost temporarily on the right side; cremasteric reflexes active; plantars slight, flexor, later lost; ciliospinal reflex present, later not obtained; pupils responded to light and accommodation, at first equal, later left slightly dilated and left palpebral fissure somewhat narrowed; retention of urine and constipation; delirium. Death on the ninth day.

*Autopsy.*—Cranial fracture; extensive extradural hæmorrhage in distribution of left middle meningeal artery. No apparent fracture or dislocation of cervical vertebrae, but the author thinks it likely that the fifth had been displaced, and then was spontaneously reduced; opposite the fourth and fifth cervical vertebrae were adherent clots of blood. Marked compression of the cord between sixth and seventh cervical segments, the cord here measuring about one-eighth of an inch; the cord was soft to pressure for an inch above and below this part; microscopically, hæmorrhage into the grey matter extending from eighth cervical segment up to, and destroying ventral portion of, posterior horn of fifth segment; otherwise, fifth cervical segment normal, as also cord above this level and below eighth cervical segment (twelfth dorsal, second and fourth lumbar segments specially examined).

The most interesting feature of this case is the evidence it affords of the location of the centre for the biceps-jerk. This is usually given as the fifth and sixth cervical segments. Potts thinks it likely, from the facts of his case, that the fifth segment has most to do with the biceps-jerk, and that the sixth plays only a subordinate part, if any. The left side of the fifth segment was not involved, and the left biceps reflex was active throughout. Probably the right side was not involved at first, and the right biceps reflex was then active; but later the hæmorrhage gradually extended upwards so as to implicate the right posterior horn, and the reflex then disappeared; while later still, as some absorption took place, the functions of this part of the segment partially returned, and the reflex was again feebly present.

Attention is also briefly drawn to other points of interest, *e.g.* the persistence of the ciliospinal, cremasteric, and plantar reflexes—the last being of the flexor type—the distribution of the motor and sensory paralysis, and the presence of extensive extra-dural cerebral hæmorrhage, with practically no symptoms except initial loss of consciousness.

A. W. MACKINTOSH.

**TUBERCULAR GENERAL PARALYSIS: PARALYTIC SYN-**  
**(220) DROME DUE TO TUBERCULAR ENCEPHALITIS.** (*Para-*  
*lysis générale tuberculeuse, etc.*) KLIPPEL, *Rev. Neur.*, April  
 15, 1905, p. 377.

KLIPPEL maintains that the symptom-complex of general paralysis may arise in three conditions due to tubercular infection: (1) Tubercular encephalitis with tubercles in the meninges or in the brain and mid-brain, accompanied by diffuse cortical degeneration. (2) Diffuse encephalitis without specific tubercular lesions in the cortex. (3) Inflammatory encephalitis due to mixed infection of pulmonary origin. In the present communication he gives the results of the pathological examination of a case of general paralysis "absolutely undeniable both in its symptoms and course." Instead of reporting the clinical observation he states that eight physicians, including MM. Babinski and Magnan, diagnosed general paralysis. The clinical phenomena mentioned are enfeeblement of the intelligence with apathy, indifference, occasional excitement, impaired memory, inequality of pupils, embarrassed speech, muscular weakness, apoplectiform attacks, transitory paralysis of speech and of limbs, increased knee-jerks, slight ankle clonus. Post-mortem the lungs showed large caseous tubercles without cavities. The meninges were neither thickened, nor adherent; no granulations on the floor of the ventricles; two tubercles in the bulb, one in the right cerebellar hemisphere, two in the left cerebral hemisphere, one of which occupied the tip of the temporal lobe, the other the optic thalamus; no neuroglia proliferation except in the neighbourhood of the tubercles; diffuse cortical changes. Klippel concludes that this case is an additional proof to convince the most sceptical that general paralysis is a syndrome and not a morbid entity. The clinical observation of the case would have been of great interest.

C. MACFIE CAMPBELL.

**ON CHRONIC PROGRESSIVE CHOREA** (*Sulla corea cronica*  
**(221) progressiva.)** G. DADDI, *Riv. di Patol. nervosa e mentale*,  
 Fasc. 4, Vol. x., 1905, p. 153.

THE writer records two cases of Huntingdon's chorea with autopsies:—

CASE I. Man æt. 65. Several of his ancestors had suffered in a similar manner, and four of his brothers and sisters had died from chorea. When admitted into hospital he showed choreic movements slightly controlled by the will; they ceased during sleep,

and the movements of the eyes were not affected. There was marked mental deterioration, the patient being partially demented. The choreic movements prevented speech and made eating difficult. He died from marasmus about a year after admission.

Examination of the brain and spinal cord showed changes by the Nissl method in the small cells and the middle-sized pyramidal cells chiefly of the superficial layer, but to some extent of the deeper layer of the cerebral cortex. The protoplasm was homogeneous, the Nissl granules disintegrated, and a large amount of yellowish pigment present. The nerve fibres were reduced in substance. Many of the nervous elements were surrounded by round, clear cells. The walls of the arterioles were somewhat thickened. These changes were most marked in the frontal and psycho-motor areas.

In the cerebellum some of the cells showed Nissl changes. No obvious alteration in the spinal cord.

CASE II. Man æt. 66. No hereditary or familial history. Up to adolescence he had suffered from epilepsy, and on one occasion had attacked the members of his family. On account of an outburst of insanity he had been arrested by the police.

Some years later, after a domestic bereavement, the choreic movements began in the neck and limbs. These incapacitated him from any work, though he was able to walk with difficulty.

Shortly after admission to hospital he attempted suicide, the mental deterioration became more marked, and the attacks of epilepsy more frequent. The disordered movement was that of a chorea, and death took place by marasmus, partly due to the difficulty in feeding the patient. The brain showed changes very similar to those observed in the previous case, and especially in the frontal and psycho-motor regions, there was also considerable increase of neuroglia. The walls of the arterioles were thickened, but no particular perivascular infiltration. Similar but less marked changes were found in the cornu ammonis. In the cerebellum, pons, and medulla, some changes in the cells were found.

The interstitial tissue of the spinal ganglia was also increased; some of the fibres in the posterior roots in the sacral region showed recent degeneration.

The writer then discusses these two cases; he points out that though heredity was wanting in the second case, yet other observers agree that this influence may be represented by other morbid conditions in the family. The association of epilepsy with chronic chorea is also known. He then considers in some detail the cause of the morbid changes found in the nervous system; points out the peculiar intensity with which they affect special parts (the frontal and psycho-motor regions) of the nervous

system, and concluding that they are not due either to senility or to primary vascular degeneration, but that the hereditary basis of the disease indicates an especial liability of the cellular elements of the neurons to undergo alteration; that the first appearance of the disease coincides with the development of mental alienation, and that the vascular and other changes are to be considered as secondary.

W. B. WARRINGTON.

**TETANY AS A COMPLICATION OF GASTRIC DILATATION,**  
(222) **WITH NOTES OF A CASE.** TRIMBLE, *Brit. Med. Journ.*,  
May 6, 1905, p. 986.

THE patient, a commercial traveller, aged 45, had suffered from attacks of torpid dyspepsia for many years, pain following about an hour after eating, and only relieved by vomiting, enormous quantities of imperfectly digested food being brought up. Tetany supervened, "the upper arms being pressed to the sides, the forearms bent at the elbows, the fingers bent at the knuckles, with the thumbs inside the palms. The feet were also contracted, and there were twitchings about the mouth." The patient was conscious during the attacks, which were preceded by "prinkling," and accompanied by severe pain, relieved by hot water bathing. Gastric lavage, the procedure which—short of operative interference for the relief of the pyloric or other obstruction causing the dilatation—is now regarded as the only effective treatment, was not adopted; delirium and hiccough supervened, and death, preceded by coma, occurred on the seventh day. The lower border of the stomach is stated to have reached below the umbilicus on the fourth day, but no record is given of its position at an earlier or later period, or during the actual attacks of tetany. Trousseau's sign was present only to a slight extent. Chvostek's sign was absent.

W. E. CARNEGIE DICKSON.

**GUMMA OF THE PITUITARY BODY, TOGETHER WITH OBSER-**  
(223) **VATIONS UPON THE OCCURRENCE OF GIANT CELLS**  
**AND TUBERCLE-FORMATION.** (Ueber ein Gummi der  
Hypophysis, nebst Bemerkungen über Riesenzellen und  
Tuberkelstrukturen.) STROEBE, *Zieglers Beiträge zur pathol.*  
*Anat. u. zur allg. Path.*, Bd. 37, Heft 3, 1905, p. 455.

STROEBE publishes this case, not so much because of its rarity and its clinical interest, as on account of the histological conditions found present in the diseased organ, more especially the occurrence of giant cells and tubercle-like miliary gummata, in addition to

the more usual necrotic and proliferative changes found in syphilitic disease of the pituitary gland. The patient, a female of 52 years, had been ill for about seven weeks previous to her admission to hospital. She was in a drowsy or semi-comatose condition, the urine was diminished in quantity, and contained a large amount of albumin, together with leucocytes and granular casts. There was marked tremor of the extremities, and a slight degree of muscular rigidity. The reflexes were readily elicited, especially the knee-jerk; there was no ankle-clonus; the pupils reacted, and were of equal size; and the fundus showed no abnormality. About a week after admission, the right pupil became dilated, and did not react to light or for accommodation, while the left pupil was smaller in size, and still exhibited a slight amount of reaction; the patient became completely comatose and died next day, the clinical diagnosis being nephritis and uræmia.

In the various organs unmistakable evidence of tertiary syphilis was found at the post-mortem examination; the pituitary was distinctly enlarged, its normal structure being almost entirely replaced by a necrotic, caseating, gummatous mass, and there being also present small cellular areas and giant cells closely resembling those found in certain tubercular conditions. Staining for the bacillus tuberculosis was, however, negative, and the author is of opinion that these structures were in reality miliary gummata and syphilitic giant cells, as has already been asserted and described by several authors, of whose cases he gives a short résumé.

W. E. CARNEGIE DICKSON.

**A CASE OF ACROMEGALY WITH ENLARGEMENT OF THE  
(224) PITUITARY, THYROID, AND SUPRARENAL GLANDS.**

(Un cas d'acromégalie avec lésions hyperplastiques du corps pituitaire, du corps thyroïde et de capsules surrénales.)

BALLET et LAIGNEL-LAVASTINE, *Nouv. Icon. de la Salpêtrière*, mars-avril 1902, p. 176.

ALTHOUGH the clinical picture of acromegaly is now well defined in its details, and the classical researches of Pierre Marie have established the connection between this disease and the part played in its causation by the pituitary gland, yet many points in the pathogenesis of acromegaly are still very obscure.

The patient whose case is described was a woman of 70 years, who showed the usual symptoms of the disease, *e.g.* enlargement of the bones and soft parts of the face and extremities, etc., together with headache, progressive deafness, pains in the limbs. Paraplegia, following a fall from a ladder, was also present. There was no sugar or albumin in the urine. The disease lasted six



years, death being preceded by coma. On post-mortem examination the pituitary gland was found to be enlarged to the size of a filbert and weighed 4 gms., the enlargement being confined to the anterior or glandular lobe of the organ, which was very soft and adherent to the dura. The sella turcica was enlarged, its vertical, frontal, and sagittal diameters measuring 15, 21, and 23 m.m. respectively, and its walls were very much thinned. The pathological condition affecting the pituitary appeared to be a very marked proliferation of the glandular tissue of the organ. Towards the surface the acinar arrangement of the cells was still preserved; the latter were mostly columnar in shape, many of them being eosinophil, and others exhibiting the iron reaction characteristic of the "siderophil cells" described by Launois and Mulon (*Soc. de biol.*, 28 mars 1903). Some of the acini contained a small mass of colloid material in their interior, whilst others showed proliferation of their cells, which were arranged in several layers. Towards the central part of the glandular lobe this proliferation became so extreme that the acini had lost their outline and had become merged in a uniform mass of proliferated epithelial cells, *i.e.* "an attempt at excessive hypertrophy of the gland, relatively successful in the cortical zone, less so in the intermediate layer, and a mere chaos of newly-formed cells and hæmorrhage at the central part with only a very faint resemblance to normal pituitary structure."

The thyroid and suprarenal glands were also enlarged, there being a hypertrophy of the glandular cells, and a sclerosis or proliferation of the fibrous tissue framework of these organs, and there being also an adenomatous condition in the case of the suprarenals. The choroid plexuses of the lateral ventricles showed a large number of calcareous bodies composed of concentric layers arranged round a central mass containing acicular crystals.

A detailed description is also given of the other organs and bones, the most interesting point brought out being that no proliferation or evidence of activity was found in the marrow of the thickened bones, and therefore, if such had been present at an earlier period, it had disappeared in the later stages of the disease.

The authors discuss the pathogenesis of acromegaly, and conclude that the hypertrophy and increased secretion of the pituitary is a necessary factor in its production, but is not in itself sufficient to produce the disease. Some perversion or aberration of the secretion is probably also necessary (*cf.* thyroid in exophthalmic goitre); as well as the various pathological conditions in the thyroid, suprarenals, and perhaps also in the choroid plexuses. We may be beginning to have some idea of the physio-pathological mechanism of acromegaly, but its actual cause is still absolutely unknown.

W. E. CARNEGIE DICKSON.

**THE LOCATION WITHIN THE SPINAL CORD OF THE FIBRES  
(225) FOR TEMPERATURE AND PAIN SENSATIONS.** W. G.

SPILLER, *Journ. of Nerv. and Ment. Dis.*, May 1905, p. 318.

SPILLER gives a short note of a case which he regards as "the best evidence that has yet been offered for the location of the fibres for temperature and pain within the tracts of Gowers." When first seen, the patient had numbness of legs below the knees; only slight weakness in the legs; sensation for touch was normal, or nearly normal, on the legs; sensation for pain was abolished, or nearly abolished; while sensation for temperature was almost lost on the legs below the knees, and much impaired over the thighs and lower part of trunk. There was slight kyphosis at about the tenth dorsal vertebra. After a fall he became completely paralysed in the legs, knee-jerks much exaggerated, Babinski's sign present; sensation for touch was impaired on the right leg, but only temporarily; sensations for pain and temperature remained much impaired on the legs. Death four months after the accident.

Tuberculous meningitis and caries of the vertebræ were found, and a small tubercle was present in the right lateral column at the extreme lower end of the dorsal region of the cord, involving the area of Gowers' tract; about one-half to one inch higher, another small tubercle was found involving the left tract of Gowers. There was some myelitis, but Spiller thinks it had chiefly developed after the fall.

ASHLEY W. MACKINTOSH.

**UNILATERAL INVERSION OF BABINSKI'S PHENOMENON IN**

**(226) POTTS' PARAPLEGIA.** (De l'inversion unilatérale du phénomène des orteils dans un cas de paraplégie pottique.) BARD, *Rev. Neurol.*, Mai 30, 1905, p. 508.

THE patient, a man 65 years old, with well advanced Potts' disease, presented a flaccid paraplegia, with brisk knee-jerks, no ankle clonus, and Babinski's phenomenon on the left only, and always; on the right there was a flexor response, very slow and deliberate, however, akin to the extensor response on the other side, except as regards the direction of the movement, and quite different from the ordinary quick flexion of the normal foot. It is noted that there was malformation of the right foot due to an accident, resulting in a pes equinus with foreshortening, the patient leaning his weight chiefly on the distal ends of the metatarsals. The autopsy revealed a caries of the 6th, 7th, and 8th dorsal vertebræ, with diffuse degeneration of the cord and obvious

pachymeningitis at the same levels. No difference was observed, on microscopical examination, between the two halves of the cord. The view, admittedly hypothetical, taken is that the deformation of the foot has led to an inversion of the ordinary relation and balance between flexors and extensors, so that had the patient been otherwise healthy he would probably have had an extensor response on the right: the medullary lesion leading in the usual way to the reversal of the normal reflexes has changed the right response from extension to flexion.

S. A. K. WILSON.

**EPILEPTOID TREPIDATION OF THE FOOT DURING ANÆSTHESIA.** (227) **THESIA.** (*La trépidation épileptoïde du pied pendant l'anesthésie.*) LANNOIS and CLEMENT, *Rev. Neurol.*, May 30, 1905, p. 511.

DURING the administration of an anæsthetic, the first reflexes to disappear are the ocular and the cutaneous. The knee-jerks are exaggerated, to disappear later.

Foot clonus is not in the same category as the knee-jerk: it commences to be brisk just after the loss of the corneal reflex, and goes on increasing up to and after the point when the patient awakes again. The exaggeration is quite independent of muscle tone. This reflex centre is possibly situated between that of the tendon reflexes and the respiratory and circulatory centres.

S. A. K. WILSON.

**A NOTE ON THE CONDITION OF THE TENDO-ACHILLIS-JERK IN DIPHTHERIA.** (228) **JERK IN DIPHTHERIA.** J. D. ROLLESTON, *Brain*, 1905, p. 68.

THE following are the conclusions arrived at by the author from an examination of the ankle-jerks in 100 cases of diphtheria until the time that the patient was allowed to sit up:—

(1) The tendo-Achillis-jerks are affected in a considerable proportion of all cases of diphtheria, though less frequently than the knee-jerks.

(2) The frequency and extent to which they are affected bear, like albuminuria and paralysis, a direct relation to the character of the initial faucial attack.

(3) They are completely abolished in all cases of diphtheritic paraplegia.

(4) Their absence may be the only evidence of loss of motor power in the lower limbs.

(5) Like the knee-jerks, they are liable to be affected at an early stage of the disease, and to remain absent after disappearance of all diphtheritic paralysis, properly so-called.

(6) Like the knee-jerks, again, they may be unequally affected on the two sides, and, like the former, they may be unusually brisk before they become sluggish and finally disappear.

(7) The Achillis-jerk, like the knee-jerk, after it has been lost, may reappear on one side before it does so on the other.

EDWIN BRAMWELL.

**A CASE OF ALEXIA WITH AUTOPSY.** EDWIN BRAMWELL,  
(229) *Scot. Med. and Surg. Journ.*, July 1905, p. 15.

PATIENT, a male aged 63, understood everything said to him, and spoke fluently and well. Very pronounced letter- and word-blindness, but with little defect in ability to recognise numerals. The only noticeable defect in written speech was occasional difficulty in writing to dictation the first letter of a word or sentence. Inability to read what he had written a few minutes before. After tracing over with his finger certain letters and words written on blackboard, was in a few instances able to decipher them, though unable to do so at sight. Right hemianopia, no object- or mind-blindness, no hemiplegia or hemianæsthesia. Memory very defective, especially for recent events. Intelligent. Visual acuity good. Right-handed.

History indefinite. Cirrhotic Bright's disease with markedly thickened arteries. Absence of general symptoms of new growth.

Death with uræmic symptoms.

*Autopsy.*—An old softening limited to distribution of left posterior cerebral artery.

Microscopic examination showed cortex of angular and supra-marginal gyri to be intact. Several photographs illustrating microscopic appearances.

EDWIN BRAMWELL.

**ON THE VALUE OF TREMOR AS A SIGN OF ALCOHOLISM.**

(230) (*Zur Berwertung des Tremors als Zeichen des Alkoholismus.*)

Professor FÜRBRINGER (Berlin), *Berliner klinische Wochenschrift*,  
May 22, 1905, p. 629.

THIS communication is based on the observation of 500 patients in hospital and private practice. Special care has been taken in sifting the cases as regards their drinking habits. They have been arranged in the two groups of abstainers or moderate drinkers and heavy drinkers. The drawing of a dividing line between the two is often difficult. Those cases whose daily amount of drink did not exceed a litre of beer or half a bottle of light wine, and who either abstained from strong spirits or only took it occasionally, were included in the first category. As

regards the degree of tremor present, the cases were grouped in three classes: (1) Those showing no tremor, or tremor barely perceptible; (2) those with moderate degree of tremor; (3) those with severe tremor. Analysing the 500 cases on these lines, the following was the result:—

Tremor absent, or nearly so, in 194 (91·5 per cent.) non-drinkers, in 18 (8·5 per cent.) drinkers.

Tremor moderate, in 153 (73 per cent.) non-drinkers, in 56 (27 per cent.) drinkers.

Tremor severe, in 28 (35·5 per cent.) non-drinkers, in 51 (64·5 per cent.) drinkers.

The author draws the following conclusions: (1) Even actual drinkers may be free of tremor; (2) hand tremor of moderate degree does not justify the drawing of conclusions as to abuse of alcohol; (3) for the diagnosis of alcoholism, marked or even very marked tremor is not by itself to be taken as giving a high probability, but as approaching that of 2 to 1. One must be cautious in making tremor by itself a basis for diagnosis. In more than half of the cases a marked tremor of the hands was present in no way connected with alcoholism.

After discussing some other aspects of tremor, the author gives an analysis of the cases according to sex:—

*Among 328 Men.*

Tremor was absent, or nearly so, in 102 non-drinkers, 17 drinkers.

„	moderate,	„	„	88	„	54	„
„	severe,	„	„	18	„	49	„

*Among 143 Women.*

Tremor was absent, or nearly so, in 76 non-drinkers, 1 drinker.

„	moderate,	„	„	52	„	2	drinkers.
„	severe,	„	„	10	„	2	„

*Among 29 Children (under 12 years of age).*

Tremor was absent, or nearly so, in 16 non-drinkers, 0 drinkers.

„	moderate,	„	„	13	„	0	„
„	severe,	„	„	0	„	0	„

Thus the male sex, with its much greater tendency to drunkenness (37 per cent.) than the female (3·5 per cent.), shows as to tremor a higher percentage (64·46 per cent.), but this relationship is reversed when we leave the drinkers out of account (32·43 per cent.).

Notwithstanding possible fallacies, intense tremor of the outstretched fingers remains one of the most characteristic symptoms of alcoholism, and must have a higher value assigned to it than Quinquad's sign.

A. HILL BUCHAN.

**PSYCHIATRY.**

- RECORDS OF THE MENTAL INVENTORY OF HEALTHY PERSONS**  
 (231) **AS A STANDARD FOR THE EXAMINATION OF DEFECTS**  
**IN THE SICK.** (Aufnahmen des geistigen Inventars Gesunder  
 als Massstab für Defektprüfungen bei Kranken.) E. RODEN-  
 WALDT (of Breslau), *Monatsschr. f. Psych. u. Neur.*, Bd. 17, p. 17.

IN the examination of the insane it is usually assumed that all persons with an ordinary school education possess a certain body of knowledge. The author put this assumption to the test by the examination of 174 apparently healthy recruits; he aimed at determining not the intellectual activity of the men, but simply their store of acquired facts, and to do so he used a series of questions from all those branches of knowledge which form part of a simple education. The results were startling, and showed that a large number of healthy individuals do not know simple facts, ignorance of which in the insane is straightway assumed to be pathological. Out of the 174 recruits only 82 said the alphabet correctly. One can not set up any definite standard of acquired general knowledge which can be used to determine a defect in the individual, and ignorance of even the simplest facts of education is not necessarily indicative of mental affection.

C. MACFIE CAMPBELL.

- CONSIDERATIONS ON THE PATHOLOGICAL ANATOMY OF**  
 (232) **DEMENTIA PRÆCOX APROPOS OF A CASE.** (Considérations sur l'anatomie pathologique de la démence précoce à propos d'un cas.) G. DOUTREBENTE and L. MARCHAND, *Rev. Neur.*, April 15, 1905, p. 386.

PATIENT was a young man who, after hardships experienced in the war of 1870-71, became depressed and developed exaggerated religious ideas. In 1874, at the age of 26, his mental status showed mental dulness, ideas of persecution, hallucinations of sight and hearing. Patient progressively deteriorated; death in 1904 from hæmoptysis. Post-mortem examination showed considerable thickening of the pia arachnoid with new formation of vessels, evidently of long standing; in places the pia was intimately united to the cortex; pigmentation and atrophy of pyramidal cells; outer cortical layer showed great neuroglia proliferation. In dementia præcox the lesions described are very varied: the disease may arise through lesions implicating the meninges and the various cortical tissues, or through lesions of the pyramidal cells alone.

C. MACFIE CAMPBELL.

**DEMENTIA PRÆCOX IS A SUBACUTE OR CHRONIC MENTAL**  
 (233) **SYNDROME OF TOXIC-INFECTIOUS ORIGIN.** (*La démence précoce est un syndrome mental toxi-infectieux subaigu ou chronique.*) M. DIDE (of Rennes), *Rev. Neur.*, April 15, 1905, p. 381.

In the hebephrenic and catatonic varieties of dementia præcox, chronic enteritis is common. The liver showed fatty degeneration in all cases examined by the author; in 15 out of 32 cases this degeneration was extreme. The decrease of urea observed in catatonia is due to hepatic insufficiency. In the paranoid form the condition of the liver is very variable. In hebephrenia and catatonia tuberculosis is very common, whereas among the other inmates of asylums tuberculosis is much rarer. Dide concludes that hebephrenia and catatonia form a toxic-infectious psychosis. He does not discuss the diagnosis of the cases upon which he bases his results.

C. MACFIE CAMPBELL.

**STUDIES ON AN ECLAMPTIC PSYCHOSIS.** (*Studien über eine*  
 (234) *eklamptische Psychose.*) K. HEILBRONNER (of Utrecht),  
*Monatssch. f. Psych. u. Neur.*, March-May 1905.

PATIENT was a peasant girl aged 18, who, when over eight months pregnant with her first child, was seized with convulsions on August 1, 1904; the urine was free from albumin until the onset of the convulsions. Labour set in, and on the death of the child artificial dilatation and extraction by the cranioclast were employed. Patient had in all 22 convulsive attacks; the last one occurred at 9 P.M., August 2; she then slowly came out of coma, became restless with wide movements of the limbs on the right side, paresis on the left. Next day she was quiet, did not believe that she was delivered. For several days she talked continuously in a slow monotonous voice, but showed great want of motor initiative; she knew she was in the clinic, was confused as to the rooms, maintained for several days that it was August 3. She became clearer on the onset of pneumonic symptoms, made a steady recovery, was discharged recovered September 30.

In discussing the case, Heilbronner calls attention to the motor phenomena—the unusual combination of loquacity with general akinesia: patient seemed to desire to initiate certain movements; there seemed no difficulty in the motor discharge, but spontaneous impulses failed to reach the site of motor discharge. She carried out movements on request, but asked nurse to remove flies bothering her; asked physician to put down her arm, which remained

suspended in the air after he had raised it; and commented on her own movements in an impersonal way. Her speech was slow and monotonous; her stream of thought showed perseveration, distractibility, and flighty associations. Perseveration was a marked symptom and was seen in many ways; she not only showed it by repetition of words or phrases, but also in the repetition of her peculiar grasp of certain objects with certain mistakes, and in responding to questions after an interval occupied with other remarks. At first patient, although showing a tendency to flight of ideas, was not distractible; when she later showed distractibility it was of a limited nature and not shown in reactions to the ordinary test objects; here, too, the perseveration showed itself in the distractibility being elicited by the same objects on different occasions.

Patient had difficulty in grasping objects and situations as a whole, unless they were of special interest to her; all the details were observed, but the combining activity was defective; hence the defective orientation, as she was unable to combine the elements she perceived into one whole. Her amnesia for the time of the psychosis was rather an inability to combine the details of her memory pictures into a definite sequence of events, but there was marked memory defect for the three or four weeks preceding the onset of convulsions.

The cases of eclamptic psychosis published have been of very diverse nature. Heilbronner calls attention to the close relation between the eclamptic and the epileptic psychoses. In his summing up of the case he associates the right-sided irritative symptoms with the talkativeness of the patient.

C. MACFIE CAMPBELL.

#### **A SPECIAL TYPE OF ERYTHROCYTE IN DEMENTIA PRECOX.**

(235) (*Di una speciale forma del globulo rosso nella demenza precoce.*) PIGHINI and PAOLI, *Riv. Speriment. di Freniatria*, 1905, Vol. xxxi., f. ii.

IN a previous article, Pighini has already described a special method of staining red blood corpuscles, which gives the following appearances. Within the outer membrane of the erythrocyte there is a zone of clear protoplasm, internal to which there is a round spongy body with a granular surface. Studied in the erythroblasts circulating in the blood of mammalian embryos, these granules are scattered throughout the whole cell, in the centre of which there is seen a chromatin nucleus. Around this nucleus there is often a clear, non-granular zone. As the erythroblast becomes older the granules gradually collect into



the centre, and finally we get the appearance already described. The writers find this appearance to be always present in the erythrocytes of normal human blood.

They have used this special method of staining in the examination of the blood of patients suffering from various forms of mental disease, *e.g.* epilepsy, dementia precox, Korsakoff's disease, general paralysis, etc., but dementia precox alone has yielded results worthy of note.

The results of the examination of ten cases are recorded in this article, along with full notes of each case and its diagnosis (hebephrenia, katatonia, etc.).

The changes found in the red blood corpuscles are briefly as follows. The central granular body takes on the stain more faintly, the volume of this body is increased, and in some cases occupies nearly the whole cell. Other erythrocytes have a clear space in the centre, within which there is a small deeply staining body, apparently the remains of a nucleus. In some cases the erythrocytes were shrivelled and folded. These changes were found in all the erythrocytes in some of the cases; in other cases there were normal erythrocytes present as well.

Other changes in the blood:—Sometimes the blood is more watery, sometimes there is a polymorphonuclear leucocytosis and an increase of blood plates.

The authors conclude that these changes do not indicate a degenerated condition of the cells, but a physiological one, and refer to the similar appearances found in embryonic mammalian blood in support of this view.

They claim that this discovery supports the theory of Kraepelin that dementia precox is due to a disturbance of bodily metabolism.

The article describes the method of staining the blood, and is accompanied by diagrams.

R. G. GORDON.

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\* A number of references to papers on Treatment are included in the Bibliography under the Individual Diseases.

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### THE FIFTEENTH CONGRESS OF FRENCH ALIENISTS AND NEUROLOGISTS.

WE have been asked to announce that the above Congress will be held this year at Rennes from the 1st to the 7th of August, under the presidency of Dr A. Giraud, Director of the Asylum of Saint-Yon (Seine-Inferieur).

The Congress is open to physicians of all nationalities.

The subjects chosen for discussion are :—

1. Psychiatry.—Hypochondria, introduced by Dr Roy of Paris.
2. Neurology.—Ascending Neuritis, by Dr Sicard of Paris.
3. Treatment.—Balneology and Hydrotherapy in the treatment of mental affections, by Dr Pailhas.

An important place is reserved for original communications on psychiatric and neurological subjects, and for the presentation of patients and specimens.

Further information regarding the Congress may be had from the Secretary, M. le Dr J. Sizaret, Rennes.

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**NOTE ERROR.**—May number, page 88, Method 5, line 11, for 24 hours read 2 to 4 hours.

# Review of Neurology and Psychiatry

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## Original Articles

### THE GROWTH OF HISTOLOGIC TECHNIQUE DURING THE NINETEENTH CENTURY.

By CLARENCE B. FARRAR,  
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THE status of knowledge of the pathologic changes in an organ depends upon the accuracy and detail of the knowledge of the normal structure of that organ. In like measure, the knowledge of an organ's normal histology is determined by the efficiency of the technique with which the observer is armed. The stages by which the science of finer brain anatomy has advanced may be followed, therefore, by simply inquiring into the origin and development of the more important technical procedures by which central tissue has been prepared for study, it being axiomatic that refinement of method and extension of knowledge go hand in hand.

In its gradual growth the histologic technique of the nervous system may be considered in three great periods :—

- I. *Primitive Period*, during which methods of examination were very rudimentary, and staining processes unknown. The period closed in 1854 with the introduction of Carmine.
- II. *Middle Period*, 1854-1884. Supremacy of the Carmine Stain.
- III. *Modern Period*, or Era of Elective Methods, inaugurated by the Fibre Stains of EXNER and WEIGERT in 1884.

## I. PRIMITIVE PERIOD.

To the primitive period belong all the coarser methods of observation, by which fresh or unstained material was examined, with or without magnification. The earliest studies were limited to the *naked-eye inspection* of the brain surface and of sections into its substance, by which were distinguished a superficial grey (*cortex*), and a central white matter (*medulla*). The former was regarded by many as the less important, if not, indeed, simply as a protecting or nutritive envelope for the delicate underlying white. The first observed fact in the anatomy of the cortex itself was the discovery by VIC D'AZYR of the intracortical white zone of the occipital region, since known as the *Stripe of Vic d'Azyr*.

For studying the make-up of nervous tissue, the method of *teasing* and, later, that of *maceration* and *isolation* were largely relied upon in preparing material for the microscope. It was assumed that the formed elements of animal tissue lay embedded in a homogeneous interstitial cement-substance (*Kittsubstanz*), and on this assumption the isolation method, either on fresh or slightly hardened material with needles or fine-pointed forceps, or on macerated tissue in which the assumed cement-substance had been dissolved by means of caustic potash, nitric acid or other reagent, naturally suggested itself as the rational method of getting closer to the nature of the elements themselves. It is easy to imagine what part these one-sided and dangerous procedures, destroying as they did structural relations *in toto*, and bringing into prominence only that portion of the assumed element which is spoken of as the cell body, or perhaps more justly expressed, that portion of the cell substance lying in immediate relation with the nucleus, may have played in establishing so securely the cell-theory as the explaining factor in the struction of the central organs. They not only practically removed the possibility of recognising assumable intercellular connections, but even made difficult the following of finer protoplasmic processes. GERLACH, in describing the minimal calibre of the axis-cylinder process at the point just proximal to the beginning of the myeline envelope, observes that in the procedure of isolation the process is easily torn off at this point,

thus accounting for the fact that the neuraxone was not earlier discovered and more generally observed.

Of the four fundamental steps of present-day technique—*hardening, embedding, sectioning* (microtome), *staining*—histologists of the early period were still halting at the first. Alcohol had long been used for its hardening and preserving qualities, although it is only in connection with the later developments of microscopic technique, during the past twenty years, that its superiority has been recognised as a fixative for the cellular components of nervous tissue.

Really the first, therefore, of the long series of hardening media<sup>1</sup> with which material was treated preparatory to further manipulation was *chromic acid*, introduced into anatomic technique by JACOBSON in 1832. This reagent, together with the chrome salts, also first used by JACOBSON, constituted the chief fixative throughout both the early and middle periods: indeed up to the time of the development of elective tinctorial methods. Other hardening agents had, to be sure, been tried, and even before the experiments of JACOBSON. KEUFFEL, as early as 1810, had used solutions of sublimate and dilute nitric acid for hardening pieces of cord. These methods did not, however, come into general acceptance, and when the chrome solutions were introduced, and their apparent superiority over alcohol in producing less visible shrinking of the tissue was realised, they were at once adopted as the hardening media *κατ' ἐξοχήν*, a distinction which they enjoyed for full fifty years. After 1859 the chrome salts were chiefly used in the solution known as *Müller's fluid*, according to the formula suggested by HEINRICH MÜLLER of Würzburg.

<sup>1</sup> *Hardening* was the original term to designate the process of increasing the consistency of fresh material as a preparatory procedure. *Fixation* is a word of later application and belongs exclusively to the modern period. It has been assumed to mean specifically the rapid transformation of soft living tissue into "fixed" dead tissue, the chief requirement of this transformation being the preservation of morphologic characters as nearly as possible *in statu quo ante*. It has, however, been pointed out by TELLYESNICZKY and others that fixation simply implies a rapid process of coagulation, which is also the essential feature of hardening, in the older sense. As a matter of fact, therefore, fixing and hardening are one and the same thing; at most, fixation might be described as the first effect of hardening. It is nevertheless convenient in modern usage to separate the two terms, inasmuch as more than one reagent often plays a part, the process of hardening being begun in one medium and completed in another, in which case the tissue is said to be *fixed* in the first solution and finally *hardened* in the second.

Material was studied unstained during the period we are considering, and various degrees of hardening were desired, chiefly to facilitate cutting, which was done free-hand with a razor or scalpel. This procedure required a manual adroitness quite unknown to histologists of the present generation.

The invention by VALENTIN of the *double knife*, which furnished tolerably thin sections of fairly definite and uniform thickness, was the first step in the improvement of sectioning methods which has culminated in the perfected microtomes of the present day. To STILLING is due further the introduction of the method of *serial sections*, making reconstruction studies possible; also the recognition of the value of *freezing* as a hardening method (1842).

In all departments of microscopic technique, the first half of the nineteenth century furnishes only the rude beginnings. The *microscope* itself was still very defective. The immersion systems and fine adjustments were unknown, and it was not until 1850 that a perfected stand was constructed by CHEVALIER in France, which has served as a model up to the present, and has made possible the development of the essential improvements of the later period, including, in addition to the higher compound objectives and the micrometer screw, the substage illuminating apparatus and the iris diaphragm.

Taking into account the coarse and uncertain methods of the early investigators as compared with the precise technique of to-day, the results which they achieved are truly remarkable. It is a sufficient corrective for any possible feeling of superiority on the part of the perfected present over the primitive past—*“zu schauen, wie vor uns ein weiser Mann gedacht, und wie wir's dann zuletzt so herrlich weit gebracht”*—to recall that all the known elements in the composition of nervous tissue, including the neurofibrils which modern histology is inclined to look upon as peculiarly its own, were recognised and described during what we have arbitrarily called the primitive period.

Nerve cells and fibres were described by EHRENBURG in 1833. Nerve fibres had already been observed by LEEWENHOEK late in the seventeenth century, by means of the rude magnifying lenses then just introduced. In 1836 VALENTIN demonstrated the wide distribution of nerve cells throughout the central

nervous system of vertebrates, and offered hypotheses as to their specific function.

SCHWANN, in 1838, gave birth to the famous cell-theory, which has dominated the biologic sciences since, reaching its climax with the dictum of WALDEYER in 1891. SCHWANN'S theory, which may be said to mark the birth of histology as a science, was simply the application to the study of animal tissue of the observation already made by the botanists that plants were composed entirely of microscopic units or plant-cells. The units or "cells" from which, according to SCHWANN, the animal economy is built up (protoblasts, KÖLLIKER), consisted of a cell-body more or less liquid, surrounded by a membrane and containing within its substance a nucleus with one or more nucleoli. The cells arose by a process of free generation from a primitive formative liquid matrix.<sup>1</sup>

The doctrine of SCHWANN was at once elaborated and improved by REMAK, KÖLLIKER, and others. Especially significant was the work of REMAK, who, while accepting SCHWANN'S definition of cells as units, pointed out the fallacy of the latter's view concerning their origin, showing that new cells arose exclusively by a process of division of the old ones. This view was further elaborated later by VIRCHOW, and summed up in his celebrated postulate—*omnis cellula e cellula* (1855).

REMAK'S description of the nerve cell (1838) was far in advance of any previously offered, and he was the first to observe the axis-cylinder of myelinated fibres, which he named "*Primitivband*," describing it, with EHRENBERG, as a hollow tube, which was occupied, however, by a number of finer structures, the primitive fibrils. Here, then, was the first observation regarding the ultimate structure of nervous tissue, made in 1838 when staining methods were unknown. Later, REMAK found that not only the axis-cylinder, but the cell processes as well, showed a fibrillar structure, and he was likewise the first to mention the complex network in the cell-body itself, made up of crossing and anastomosing fibrils entering from the processes. Like many another new and remarkable discovery, REMAK'S

<sup>1</sup> "Es ist eine structurlose Substanz da, die nach ihrer chemischen Beschaffenheit und nach dem Grade ihrer Vitalität mehr oder weniger die Fähigkeit in sich trägt, die Entstehung von Zellen zu veranlassen." [Cited by STRICKER.]

observations concerning the primitive fibrils received scant credit, and when MAX SCHULTZE in 1868 maintained anew the fibrillar make-up of axis-cylinder and ganglion cell, his views met with quite as intense opposition as that which had greeted REMAK thirty years before.

Further, the relation between nerve cell and fibre, so thoroughly demonstrated by the GOLGI method in the 'seventies, was nevertheless not a new discovery. During the primitive period, not only were cells and fibres recognised as the two chief elements in the nervous system, but the connection existing between ganglion cell and myelinated nerve fibre was also established (HANNOVER, 1840 ; WILL, 1844 ; ROBIN, 1846 ; RUDOLPH WAGNER, 1846).

Finally is to be mentioned the discovery by VIRCHOW in 1846 of the specific supporting substance of the central nervous system, to which later he gave the name "neuroglia"; and although the exact nature and distribution of the glia remained obscure up to the time of WEIGERT's *bahnbrechende Arbeit* (1895), yet the observations and deductions of VIRCHOW, as WEIGERT particularly points out, were little less than marvellous, made as they were in the period when staining had not yet become a technical procedure. Even glia fibres were seen by VIRCHOW in a case of Tabes, in material hardened simply in chromic acid.

The foregoing are the more important facts in neurohistology accumulated during the first period of histologic technique, embracing roughly the first half of the nineteenth century.

## II. MIDDLE PERIOD.

The second period includes the succeeding thirty years, and begins with the advent of staining methods. *Carmin*, the introduction of which marks the commencement of this important epoch, was earliest used on animal tissue by CORTI, but came first into general use a few years later through GERLACH (1854). Not only was carmine the initial stain of the period, it was practically the only one. To the men of that day a successful carmine preparation stood so far above the unstained material to which they had been accustomed, and furnished the key to so many possibilities of research, that for the time it seemed all-sufficient. Years were required to develop and ex-

haust these possibilities and demonstrate the need of more refined tinctorial methods.

"Jeder," wrote WEIGERT, "der die Anfangszeiten der histologischen Technik noch einigermaßen miterlebt hat, wird sich des Erstaunens erinnern, welches die ersten Karminbilder des Centralnervensystems, die er zu sehen Gelegenheit hatte, bei ihm erregten. Ich selbst erinnere mich noch sehr wohl an die begeisterten Worte, mit denen DU BOIS - REYMOND uns die Copie eines von CLARKE herrührenden Bildes eines Rückenmarksquerschnittes, der mit Karmin gefärbt war, vorführte, und wir alle hatten wohl die Idee, dass es doch etwas Grosses sein müsste, ein solches Präparat anfertigen zu können."

The new stain soon became the universal one. It gave particularly good results with chromated tissue, and, unlike the silver impregnation method, which had already been introduced by KRAUSE (1844), or the gold method, which was discovered later (COHNHEIM, 1867; GERLACH, 1868), the carmine stain did service not only for the anatomist, but to a certain extent for the pathologist as well.

The workers of the early period, and those of the first half of the second, had concerned themselves with the morphologic characters of the constituent elements of nervous tissue. The time was not yet ripe for the study of the grey and white as a whole—as organs whose composing elements were to be appreciated only in their proper places and relations.

DEITERS had drawn the universal distinction between axis-cylinder process and dendrites, which he named respectively "nerve-process" and "protoplasma-processes," the differential features of which he had described. GERLACH, working with the gold method, had created a sensation by postulating a true intercellular reticulum formed by intermingling and anastomosis of attenuated ganglion cell processes. SCHULTZE had demonstrated in fresh material treated with iodised serum (amniotic fluid of sheep or cow + iodine), the elementary structures in axis-cylinder and cell-body, and to which he gave the name "*Nervenprimitivfibrillen*."

These and other investigators were working on the *components* of nervous tissue; the *ensemble* of the central organs had not been attacked until MEYNERT declared that all nerve cells were alike in structure, function, and importance, and that what-



ever specific activity they might possess depended solely upon the manner of their connection with other cells or centres in the cerebro-spinal axis, with the sense organs, or with the peripheral musculature. With the work of MEYNERT, during the late 'sixties and early 'seventies, emphasising the importance of studying the *connections* between different parts of the brain rather than the structural characteristics of the individual elements, fibre anatomy had its birth, and soon overshadowed completely the cytohistologic studies which had gone before.

This turn in events is to be explained, in part at least, by the fact that carmine, the stain of the period, gave much better pictures of the white matter, with its simpler structure, than of the complex grey. Bundles of myelinated nerve fibres could thus readily be traced, and numerous investigators following MEYNERT'S lead turned their attention, during the latter half of the second period, to working out the various fibre tracts. In MEYNERT'S opinion, not only the important anatomic questions, but also those in neuropathology, were to be approached from the view-point of fibre anatomy. During this time, therefore, the determining of gross relations and connections, rather than of histologic details, occupied the foreground of research; and for these purposes the carmine method was destined to be fruitful of results.

In cortical studies, on the other hand, the limitations of a diffuse or indifferent stain such as carmine soon became apparent. Here was a tissue made up of a great variety of structures functionally and morphologically different. In carmine preparations all these structures were stained together in but slightly varying shades of red, axis-cylinders, ganglion cells, protoplasmic processes, neuroglia, mesodermal elements, ground substance—all took up the dye; cells were obscured by a forest of fibres, and fibres in turn by a sea of cells. Often enough, indeed, the picture offered but little which the practised eye had not already seen in unstained material; while, for revealing finer histopathologic changes, the carmine method was from first to last inadequate. It was this need particularly which called forth efforts which finally revolutionised microscopic technique, ushering in the third or modern era, in which the doctrine of specialisation is introduced into laboratory procedure in the form of elective staining methods.

Meanwhile, the necessity of thinner and larger sections than could be cut by the hand, and of serial sections of known and uniform thickness, led, during the 'seventies, to the development of the *microtome*, which has now become the chief piece of artillery in every laboratory. To facilitate cutting material, which had become more or less brittle through the process of hardening (chrome salts were still the common fixative), various inclusion or embedding operations were devised. As early as 1869, KLEBS had employed *paraffin* in a simple inclusion process, in which the molten paraffin was merely poured around the block to be cut. Later came the perfected embedding method. *Celloidin*, the other great embedding medium of later technique, was first used by SCHIEFFERDECKER in 1882. Collodion, however, which is chemically identical with celloidin but of less constant purity, had already been employed by LATTEUX for inclusion, and later by DUVAL (1879) as an embedding medium.

Although late in the second period the uncertainty of results with the carmine method had given rise to various modifications and substitutions, none of these was of sufficient importance to supplant the older process—chrome hardening and carmine staining—which remained dominant practically alone throughout the period, giving way finally to the multiple elective procedures of the present. Carmine is to-day little more than a name in history.

### III. MODERN PERIOD.

The latest period in microscopic methodique began in 1884 with WEIGERT's publication of his medullated fibre stain. All the important tinctorial methods of which the brain anatomist today makes use have been discovered and developed within this period, and have entirely superseded those previously in vogue. At present, instead of a single universal stain for nervous tissue, white and grey, one has at command a variety of specific processes, by each of which certain definite constituents of the tissue for which the process is said to be "elective" can be brought into prominence, to the exclusion, more or less complete, of all other elements, which in turn are rendered conspicuous by other appropriate *elective methods*. These several selective procedures, therefore, supplement or complement each other, and,

taken together, furnish a composite histologic picture, which, though still far from complete, is yet a great way beyond the dreams which any diffuse stain could awaken. While, then, three decades of carmine contributed more to gross brain anatomy than had been learned in all the previous years, two decades of elective methods have quite overtopped both the preceding epochs, together in the wealth and accuracy of details which they have supplied, both in fibre anatomy and in cellular and intercellular histology.

CARL WEIGERT, in working out his neuroglia stain (1895), set down the following qualifications which the method, in order to be completely successful, should possess :—

1. The stain must be elective. It must leave unstained all those structures which might in any way be confused with or obscure the elements which it is desired to demonstrate. Thus an axis-cylinder stain, to be satisfactory, must not stain glia fibres; and, on the other hand, a successful glia method must leave entirely unstained dendritic ramifications and neuro-fibrils.
2. The method must be certain. It must reveal in a constant manner in each properly prepared specimen all the structures for which the stain is elective. WEIGERT insists particularly that any method must be rejected in which the result is prejudiced by minute variations in time of any of the manipulations.
3. It is desirable that other elements for which the method is not specific shall be so far outlined by the stain as to make orientation possible.
4. The colouring of the elements electively stained should be sufficiently intense to allow their easy recognition and study.
5. The method should require as little time as possible for its accomplishment; nevertheless, the *tuto* must be considered before the *cito et jucunde*.
6. It is requisite that the various manipulations called for by the process shall not injure the specimen by making it brittle, causing undue shrinking, etc.
7. It is desirable that the preparations should have a certain degree of permanence.

These were the standards by which WEIGERT judged his method, and they serve equally well as guiding principles for all the modern staining processes. An elective stain in the strictest sense, which should present only certain definite elements of similar nature, leaving everything else totally invisible, would not be desirable for histologic purposes. It would, to be sure, make possible the study of isolated elements under ideal conditions, but would, nevertheless, by removing all possibility of orientation, largely defeat the purpose of any satisfactory method, namely, the demonstration of individual tissue constituents in their relations to each other.

In defining the essentials of the elective glia stain, WEIGERT made a statement which, while strictly true, was yet unfortunate in its results. He said: "For the topographic study of the neuroglia, all methods are to be rejected by which the fibres appear as actual processes of DEITER's cells." This statement is indeed in harmony with WEIGERT's definition of the neuroglia, by which he understood exclusively the independent intercellular fibres. It indicated, moreover, a healthy reaction against the conception of the glia, based on GOLGI pictures, in which a differentiation between fibre and cell protoplasm was impossible. The stringent requirement, however, of a differential method which should leave the bodies of DEITER's cells unstained, led to the erroneous view that the fibres were wholly independent, that their apparent connection with cell bodies was due to an error in technique, and that their only relation to the neuroglia nuclei was a spatial one. In accordance with our present knowledge, the statement of WEIGERT would perhaps be modified as follows: For the topographic study of the glia fibres all methods are to be rejected which do not reveal the fibres differentiated from the protoplasm of the cell body or its processes, whether still in connection with the cell protoplasm or separated from it.

An elective ganglion cell stain we do not possess; yet this is not a very serious defect, inasmuch as the various cell methods now in use reveal so admirably both external and internal morphology of the several cell types that a confusion of ganglion cells with other elements in normal tissue is rarely possible. EDINGER nevertheless observes (1904), just as did GERLACH, working with the carmine method thirty-five years ago, that it is not always possible to distinguish certainly between

glia and ganglion cells. EDINGER says: "It appears much easier to say what is glia in diseased tissue than in normal." As a rule, the reverse of this statement would appear to be true. Given normal ganglion cells and normal glia cells, each possesses distinguishing characters which suffice in all but the rarest instances for a differential diagnosis, whereas under certain pathologic conditions these differential characters may be more or less lost, and the difficulty of distinguishing between the two cell forms correspondingly increased. This is particularly true in the case of certain regressive changes in the smaller nerve cells, which may thus become scarcely recognisable as such.

The methylene blue method of NISSL, and other similar procedures, have been spoken of as elective nerve cell stains. This, of course, they are not. They are merely elective *cell* stains, for they reveal with equal facility all nuclei, and those portions of the cell protoplasm immediately surrounding, in both nervous and non-nervous elements, which in the given case would be visible by any method. Normally, for example, endothelial cell bodies are invisible, or practically so; glia cell bodies, while visible, are usually insignificant, but the abundant chromophilic substance of the nerve cell protoplasm makes these elements conspicuous above all others. In pathologic tissue, however, in which endothelial or glia cells have undergone progressive changes, these constituents present well-stained cell bodies often of considerable dimensions.

While a method which should reveal exclusively ganglion cell bodies or glia cell bodies with their nuclei, would doubtless clear up more quickly and certainly than is at present possible, certain points in the histopathology of these elements, yet the demand for such a method is certainly much less than was that for the various other elective processes which have been the outgrowth of the modern period.

The certainty and constancy of a method do not require that the elements shall appear *in statu quo ante mortem*. This is not only obviously impossible, but thoroughly non-essential. That in any finished microscopic preparation, the elements appear more or less as artefacts, does not necessarily detract from the value of the method or influence of the study of such preparations. The object is not to see how, for example, a *living* nerve cell looks, but to obtain a reproduction of the nerve cell which, under

determinate *post mortem* conditions and with uniform technique, shall be so constant that variations in its appearance can be associated with *ante mortem* causes—in other words, to obtain the “equivalent picture” of the cell in the sense of NISSL.<sup>1</sup>

The elective procedures of the modern epoch fall into four categories in the order of their development, as follows:—

- (A) Myelinated nerve fibre methods.
- (B) Elective cell stains (including both nervous and non-nervous elements).
- (C) Neuroglia fibre stains.
- (D) Methods for neurofibrils.

(A) *Myelinated Nerve Fibres.*

During the carmine period the white substance of cord and brain with its myelinated nerve fibres was the chief object of study, and it was to demonstrate just these structures in a more complete and certain manner than had been possible with carmine, that the experiments were made which resulted in the first genuine elective stain—that of the myeline sheath.

Although to WEIGERT is due the credit of having elaborated a conveniently available and trustworthy sheath stain, which to-day, either according to the author's description or in some of its modifications, is the universal method for myelinated fibres, he was not the first to show that these elements could be satisfactorily demonstrated by staining their myeline envelope instead of the axis-cylinder, which was the part revealed by the carmine method. This was EXNER's discovery. The method of EXNER (hardening in osmic acid, sections treated with ammoniated glycerine) is at present chiefly of interest in having furnished the earliest differentiation of the myeline sheath, and in having been used by TUCZEK (1884) in his well-known studies on the disappearance of cortical fibres in dementia paralytica. On account of the uncertainty of the method and lack of per-

<sup>1</sup> “Unter Nervenzellenäquivalent verstehen wir demnach das mikroskopische Strukturbild der im Gewebe vorhandenen Nervenzellen des in einer bestimmten Weise getödteten Thieres, das bei einer bestimmten mikroskopisch-technischen Behandlung des Nervengewebes unter bestimmten Voraussetzungen erfahrungsgemäss mit einer gesetzmässigen Gleichheit zur Darstellung gebracht werden kann.”—NISSL, *Encyclopädie der mikroskopischen Technik*, Bd. ii. S. 988.

manence of the preparations, EXNER's technique did not come into common use.

WEIGERT's method was developing contemporaneously with the work of EXNER and TUCZEK and was published in 1884 with all the essential steps, except that of the second mordant, copper, which was added the next year. Other improvements and modifications were also suggested later from time to time. Describing his method in its essentially complete form, WEIGERT wrote :—

“Jetzt endlich (1885) nach dreijähriger unausgesetzter Arbeit war ich insofern ans Ziel gelangt, als ich auch die feinsten Fasern (*i.e.* those of the supraradiary zone) zur klaren Darstellung bringen konnte. Es waren ja noch mancherlei Wünsche zu erfüllen, aber diese Wünsche waren sekundärer Natur. Die Hauptsache war eben doch, dass nunmehr zum erstenmale nachgewiesen war, dass mit Hilfe einer Färbemethode die allerfeinsten Markfasern des Centralnervensystems dargestellt werden konnten.”

The final method of 1884-5, embodying the following four procedures—

- (1) Hardening—primary mordant (kali bichromat potassium);
- (2) Secondary mordant (copper);
- (3) Overstaining (hæmatoxylin);
- (4) Differentiation;

was the third made public by WEIGERT during the series of experiments. In the first (1882), he had used acid fuchsin as the stain, differentiating with alkaline (K.) alcohol. This was the first use of acid fuchsin in microscopic technique. In the second method (1883), basic fuchsin was employed, differentiation being accomplished with hydrochloric acid. In the third, finally (1884), hæmatoxylin (introduced by WALDEYER, 1863) was tried and found good. The “ripening” of the hæmatoxylin solution (oxydation of hæmatoxylin to hæmatein), which had empirically been found to be essential, and which had been effected by allowing the solution to stand, was hastened by the addition of an alkali (Lith. Carb.).

Throughout the experiments WEIGERT had used tissue which had been hardened and mordanted in potassium bichromate. To hasten this process he first suggested that hardening should take place in the oven; later it was found that this end

was favoured by the addition of fluorchrome to the bichromate solution (1897). By this means the originally long mordanting process could be reduced for small pieces of tissue to four to five days or a week. As a secondary or reinforcing mordant, the original copper solution was finally replaced by the one employed by WEIGERT in his glia method (1895).

As a differentiating fluid in which the sections, overstained with hæmatoxylin, were bleached, WEIGERT always preferred his original solution of iron-potassium cyanide and borax. The differentiation of PAL (first suggested by LUSTGARTEN) with potassium permanganate and oxalic acid, is criticised as less certain and trustworthy on the ground that the period during which the differentiation takes place is so short that it is difficult always to stop the process at the right moment. Decolorisation may thus easily be carried too far, in which case many of the finer fibres will have been differentiated out of sight.

A further important modification of his method lay in the substitution of the iron-hæmatoxylin mixture<sup>1</sup> introduced by WEIGERT for the older hæmatoxylin solution. By this means beautiful contrasts are obtained without danger of over-differentiation. The picture is comparable with a well done PAL, and the results, according to WEIGERT, are more trustworthy than with the latter method.

Three important facts were established by WEIGERT early during his experiments with the new method, viz.:—

- (1) The tinctorial reaction takes place in the myelin sheath itself—a fact which had not been recognised by EXNER.
- (2) The reaction is not a direct union of the dye with the myeline, but an indirect one by way of an intermedium or mordant (*amboceptor*), which forms a permanent metallic union with the myeline, and with which in turn the stain (*complement*) combines.
- (3) Not the whole substance of the sheath, but only certain of its constituents (Protagon, WLASSAK) are involved in the process.

<sup>1</sup> Equal parts of—

(A) 1 per cent. solution of hæmatoxylin in 96 per cent. alcohol.

(B) 4 per cent. aqueous solution of liquor ferri sesquichlor.

(*To be continued.*)



**NOTE ON A CASE OF ADVANCED CARCINOMA UTERI  
WITH SOME SYMPTOMS OF BULBAR PALSY  
AND ALMOST NEGATIVE MICROSCOPICAL  
FINDINGS.**

By W. B. WARRINGTON, M.D., F.R.C.P., Liverpool.

I AM indebted to the kindness of Dr Arthur Wallace for the opportunity of studying this case.

The patient, æt. 45, was admitted to the Hospital for Women in December 1903, and presented the usual signs of carcinoma cervicis uteri. Examination showed this to be inoperable, but as much as possible of the growth was removed by the curette.

A week after the operation severe headache was complained of, speedily followed by dimness of vision of the right eye, with photophobia and some prominence of the eyeball. Later external squint appeared. The tongue when protruded pointed to the left, and there was also difficulty in swallowing and articulation.

On admission into my ward on the 19th January 1904, the patient was found to have complete paralysis of the right third nerve and marked wasting, with immobility of tongue, so that articulation was impossible. The muscles forming the floor of the mouth could not be felt to contract on swallowing, and there was slight weakness of the lower part of the face. The vocal cords and palate moved normally, and no disturbance could be detected in the functions of the other cranial nerves or in the rest of the nervous system. The patient died in five days, gradually passing into coma.

So far as her condition would permit, examination showed no disease in the viscera, except the uterus, beyond a small amount of albuminuria.

A post-mortem limited to the brain was made. There was found to be considerable œdema, excess of fluid over the cerebral surface and shrinkage of the brain mass, but no gross lesion either in the meninges or brain itself. The right cavernous sinus was found filled with a soft pale clot, firmer and paler towards the wall of the vessel than in the centre.

This is the condition known as marantic thrombosis, and to it may be attributed the palsy of the eye muscles. Micro-

scopical examination by the Nissl method of the nucleus of origin and of the nerve by the Marchi method before it had entered the cavernous sinus showed these to be normal.

The paralysis of the tongue remained to be explained. The nuclei of the hypoglossal, of the facial, and of the motor dorsal and ventral (n. ambiguus) of the vago-spinal nerve were examined, and the brain stem with the pia mater examined for carcinoma cells.

No form of metastasis was found to account for the tongue palsy. Carcinoma of the brain is usually secondary and is rare, but when it occurs presents the peculiarity that the secondary growths are often extremely numerous and very small, often only being recognisable by the microscope. Hence the necessity for careful examination for points of carcinoma cells.

The only change found in the nuclei examined was in the hypoglossal, and here the majority of the cells appeared normal, yet a few showed chromatolysis.

Transverse sections of the filaments of the nerve showed by the Marchi method scattered degenerative black granules, entirely absent from the third nerve treated with the same fluids.

During the patient's life I was at a loss to explain the symptoms, which were obviously not those of ordinary bulbar palsy, and rather expected to find some carcinomatous metastasis.

I find Weisenburg (1) has recently written on this subject and collected the recorded cases, which are very few and may be briefly mentioned :—

Spiller and Weisenburg (2)—vertebral carcinoma—paraplegia dolorosa. “The muscles of the face are extremely atrophied, the lips are very thin, the tongue is atrophied on each side and is protruded very slightly beyond the line of the teeth. There are no fibrillary tremors of the face or tongue. The fifth and seventh nerves are normal.”

The necropsy was made by Dr Flexner, and the microscopical examination by the writers. There were found two largish metastases in the skull, but not penetrating into the dura, and carcinomatous tissue was not seen within this membrane. “The macroscopic examination of the medulla oblongata

revealed nothing, but in the microscopic examination some of the cells of the hypoglossus nuclei were found distinctly diseased. The nuclei of some of the cells were displaced towards the periphery, the chromophilic elements were disintegrated, and in some of the cells there was an excess of yellow pigment. We were not able to detect any implication of the hypoglossal nerves in a carcinomatous mass, and it is probable that the alteration of the hypoglossal nuclei was caused by some poisonous products of the numerous carcinomata."

Bruns (3), also one of secondary vertebral carcinoma. There was right-sided atrophy and paralysis of the tongue; right-sided paralysis of the soft palate, difficulty in swallowing, increase in the pulse-rate, and vomiting. The atrophy and paralysis of the tongue became bilateral. The macroscopic examination gave no explanation of the bulbar symptoms. Microscopic examination does not appear to have been made.

Weisenburg (1), woman, aet. 59, cancer of the breast removed thirteen years ago—recurrence in the breast and glands. There was difficulty in swallowing and in articulation—tongue, masseters, and facial muscles acted normally.

The nerve cells of the paracentral lobule of each side of the cerebellum, and the nerve cells of the nuclei of the 6th, 7th, 9th, 10th, and 12th nerves were examined by the thionin stain.

A change was found in some of the cells of n. ambiguus, and of the dorsal vagus nucleus, and of the glosso-pharyngeal nucleus; also some of the cells of the 6th and 7th nuclei. The pial covering showed no carcinoma cells.

In agreement with Weisenburg, I can only regard these conditions as due to a toxæmia.

Alfred Saenger (4) has criticised this view. He classifies the cerebral symptoms found in carcinoma elsewhere as follows:—

1. General symptoms, such as headache, loss of consciousness, apathy, coma, dizziness, noises in the ears, and vomiting. In these no changes on microscopic examination may be found.

## 2. Special symptoms.

- ( $\alpha$ ) Without microscopic change, but on thorough microscopical examination metastases of carcinoma cells found.
- ( $\beta$ ) With microscopical changes—
  - ( $\alpha$ ) Tumour growths of various sizes.
  - ( $\beta$ ) Softening or hæmorrhages without metastases.

To this classification must be added cases similar to those of Parhon (5) and Hudovernig (6), in which destruction of an organ such as the tongue (Parhon) has given rise to the *reaction à distance*, and those similar to mine due to an intoxication, in which all the changes mentioned by Saenger were absent.

If the cases quoted can be advanced as valid evidence for this view, the suggestion of Oppenheim (7) is of much interest. On the one hand, true bulbar palsy, with gross changes in the cells, and its hardly discernible causation; on the other, myasthenia, with scanty alteration in the cells and the presence of lymphoid masses of cells infiltrating the muscles, and sometimes the mediastinum.

Even were such changes constant, I cannot think that the symptoms of myasthenia are to be explained merely by the presence of cellular infiltration interfering with muscular action, and in the cases quoted there is some evidence that a toxæmia can cause profound changes in nerve cells, and yet that these changes are revealed in only a slight degree by microscopical examination.

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## Abstracts

### ANATOMY.

**ON FAMILY LIKENESSES AFFECTING THE FISSURES OF**  
 (236) **THE HUMAN CEREBRUM.** (Über Familienähnlichkeiten  
 an den Grosshirnfurchen des Menschen.) J. P. KARPLUS,  
*Arb. aus d. neurolog. Inst. in Wien*, xiii., 1905, p. 1.

WITH observations on the disposition of fissures in a series of brains taken from groups of relatives (mother and child, grandmother and uncle, brother and sister, twins, father and children), Dr Karplus has framed a highly instructive contribution to the anatomical and pathological manifestations of heredity.

A fund of details—details which cannot be epitomised—constitutes the main worth of the paper, and it is illustrated with twenty admirable plates.

The thoughtful care bestowed on the work compels acceptance of the fundamental statement that the fissural plan of the cerebrum is susceptible to the imprint of heredity: it appears that at times the whole plan is copied, that at others sulcal variations are repeated in several members of one family, and that the repetitions always affect homolateral hemispheres. In the case of twins of different sexes it is maintained, contrary to Rüdinger, that intra-uterine fissural development does not begin earlier and advance more rapidly in males than in females, but contemporaneously and equally in both.

Having prospected, proved the fertility of, and already derived much gain from this field, Dr Karplus proposes next to push his research along histological and comparative anatomical lines.

ALFRED W. CAMPBELL.

### PHYSIOLOGY.

**EXPERIMENTAL OBSERVATIONS ON THE VASCULAR NUTRI-**  
 (237) **TION OF PERIPHERAL NERVES.** (Experimentelle  
 Untersuchungen über die vasculäre Trophik der peripheren  
 Nerven.) EIKITSCHI OKADA, *Arb. aus d. neurolog. Inst.*  
*in Wien*, 1905, p. 69.

OKADA, a Tokio observer, ligatured the inferior gluteal artery above the point where it gives off the nutrient artery to the

sciatic nerve (*arteria comitans nervi ischiadici*). In this way the nerve was deprived of its chief vascular supply. As a result he found degeneration, more or less complete, and of a Wallerian type, in the peripheral portion of the nerve. As in Wallerian degeneration, there was proliferation of the nuclei of Schwann's sheath, and of the endoneural connective tissue. He concludes from this that for the nutrition of the peripheral nerves two factors are necessary, one trophic influence exerted by the corresponding ganglion cells, and another exerted by the arteries of the nerve. Neither of these alone is sufficient to maintain the nerve in proper nutrition. Both factors are necessary.

The writer, on the subject of nerve-regeneration, says that "so far as he dares to draw conclusions" from his preparations, he thinks that the old view is correct that the new fibres develop by a process of outgrowth from the central fibres, which are in connection with the ganglion cells. No arguments, however, are adduced in support of this view, which is in contradiction to the results obtained by Kennedy, Ballance and Stewart, Bethe, and others.

PURVES STEWART.

**STUDIES ON THE MAINTENANCE OF THE EQUILIBRIUM OF**  
(238) **MOTION AND ITS DISTURBANCES, SO-CALLED "FORCED**  
**MOVEMENTS."** L. J. J. MUSKENS, *Journal of Physiology*,  
xxxi., 3 and 4, June 30, 1904.

IN vertebrates with horizontal posture we can distinguish two principal types of forced movement, viz.: (1) *rolling movement*, i.e. locomotion (rotation) in a plane vertical to the longitudinal axis; (2) *circus movement*, locomotion in a circle in a horizontal plane corresponding with the longitudinal axis of the body. There is a third type of forced movement, viz., locomotion in a plane corresponding with the longitudinal axis, and vertical to the horizontal plane. In its incomplete forms (falling backwards or forwards, with analogous phenomena in the motion of the eyeballs) it is met with in the higher vertebrates after lesions in the middle lobe of the cerebellum and in the mesencephalon.

The two principal types of forced movement manifest themselves in actual movements of rotation, frequently interrupted by intervals of rest, during which trunk, head, and eyes are kept in a peculiar position characteristic for particular forced movements. There may not, however, be any actual movement, but only the characteristic attitude. As a rule they are of a passing character: they tend to disappear or to become compensated. Paralysis of muscles or groups of muscles is not the cause, nor does it accompany

any of the manifestations of the forced movements, although paralysis occasionally may be brought about by the lesion which has produced the latter.

These forced movements are common phenomena after any lesion of the cerebro-spinal axis proximal to the exit of the eighth nerve, but knowledge of them acquired from lower vertebrates must be applied with caution to beings with erect posture. In the author's experiments on *selachians* (*acanthias*, *scyllium*), hemisection of the cerebro-spinal axis through the mesencephalon (corresponding to the corpora quadrigemina of higher vertebrates) constantly produced circus movements towards the uninjured side. Hemisection of the medulla oblongata, proximally to the exit of the eighth nerve, produced with the same constancy rolling movements towards the injured side.

Numerous experiments in *frogs* bore out the same facts. After right unilateral lesion high up in the medulla, the screw-like position of the body and limbs is very marked; the left eye-ball bulges, the right pupil is smaller, the right corneal reflex absent; the right foreleg is adducted, the left abducted; the right hind leg is drawn up, the left extended; the head is rotated along the longitudinal axis towards the right side, and also bent to the right side. The close relation of the two types of forced movement is readily seen: the frogs jump to the left, and come down on the right flank.

In *rabbits* and *cats* a puncture was made with a long steel needle, applied 2 to 3 mm. posterior and lateral to the external occipital protuberance, piercing the middle peduncle. The needle was thrust vertically to the base of the skull, in a slightly forward direction. The results may be briefly recorded that "unilateral lesions of the middle and upper cerebellar peduncle, and unilateral (partial rather than complete) removal of a lateral lobe of the cerebellum cause rolling movements to the side of the lesion, or tendency to it, *i.e.* forced position in the same sense." The most vehement rolling was observed both in the cat and in the rabbit after lesion or section of the middle peduncle and the tuberculum acusticum. The rolling movements to the impaired side were often associated with circus movements to the unimpaired side.

The complex changes in the mobility of the eyeballs deserve fuller investigation:—

1. After section of the right middle peduncle in rabbits, the right eye is deviated down and forward, the left eye up and backward ("skew deviation").

2. There is found constantly (*cats*) a rotation of the eyeballs around the visual axes in such a way that the lower portion of the vertical meridian deviates to the left, the upper portion to the right, a rotation therefore in the same sense as the rolling movements.

3. In the cat, further, the rotatory nystagmus of the days after operation is synchronous in the two sides, while the direction of the quick jerk of the nystagmus is directed contrariwise to the deviation above described, *i.e.* the right eye will jerk up and to the left, the left eye downward and to the right.

After lesions higher up than the optic thalamus, rolling movements or allied forced positions are very rare, whereas temporary circus movements with conjugate deviation of head and eyes to the same side are a very common occurrence. The view the author takes of these phenomena is that the complicated act of locomotion is dependent on a system of nerve tracts and centres, asymmetrical lesions in which produce forced movements. It is probable that the efferent part of this system has nothing directly to do with the pyramidal system. The importance of the semi-circular canals, vestibular nerves, perhaps also eyesight and sense of touch, as representing the afferent side, is admitted.

S. A. K. WILSON.

**CRITICO-EXPERIMENTAL CONTRIBUTION TO THE STUDY OF  
(239) THE SUBJECTIVE PHENOMENA OF FATIGUE IN  
VOLUNTARY WORK.** (*Contributo critico-sperimentale allo  
studio dei fenomeni soggettivi di fatica nel lavoro volontario.*)  
Z. TREVES, *Riv. di Patol. nerv. e ment.*, May 1905, p. 201.

IN this interesting and important paper, Dr Treves gives a résumé of his analysis of the ergographic curve, and a comparison of his results with the results of other authors with respect to mental work, in the course of which he succeeds in establishing an interesting parallel between the two modes of work.

A diagram accompanies the paper, giving two examples of the graphs of the four factors which the author has been able to distinguish in his experiments on voluntary muscular work. These curves represent the Effort, the Work done, the Acceleration, and the Energy of Contraction. For a full account of the methods employed in distinguishing these factors and details of the experiments, the reader is referred to papers which have appeared in the *Archives di Fisiologia*, and which are doubtless known to those in this country who are following the course of this investigation. A detailed examination and comparison of these curves leads the author to conclude that the curve of Acceleration represents something above the merely nervo-muscular mechanism, that it is, in fact, the direct emanation of that psychic factor which is the unfailing concomitant of even the simplest voluntary act.

The second part of the paper is devoted to a summary and criticism of Kraepelin's analysis of the curve of mental work.



This curve is, of course, due to the interaction of many factors, and Kraepelin has attempted to resolve the complex curve into its several components. Thus he gives a graphic representation, showing how much influence is to be attributed to Practice, Fatigue, Short Pauses, Habit, accentuation of Volitional Tension (*Antrieb*), etc. Short pauses have a favourable effect on the curve, but this effect does not increase in proportion to the length of the pause, as one might expect; there is found to be an *optimum* pause. This factor Dr Treves identifies with the *Anregung* of Amberg; it consists in a predisposition to work, and can be detected after the beneficial effects of practice have had time to disappear.

Dr Treves criticises Kraepelin's analysis on several grounds.

In the first place he questions, not only the numerical values, to which apparently a merely tentative value is assigned by Kraepelin himself, but in some instances the very type of curve. For example, the curve of work influenced by Fatigue alone is, according to Kraepelin, a straight line descending rapidly to the abscissa; but Dr Treves points out that this contrasts with the hyperbolic character of all the analogous curves hitherto recognised by physiology.

In the second place, he thinks it impossible to separate the effects of Practice from those of Fatigue. When we speak of the effects of Practice, he says, in contrast with those of Fatigue, we can only refer to the complex of phenomena of the anabolic phase which succeed not only in counteracting, but in more than counteracting, the depression arising from the catabolic phase; and these two antagonistic factors, though they can indeed be separated in thought, are, from the point of view of the production of work, "two absolutely inseparable factors of the same phenomenon."

Thirdly, he maintains that the effects of Habit belong to the same order of phenomena as those of the *Anregung*. In short periods of work the improvement ascribed to Habit must obviously simply be due to the gradual adaptation of the individual to the conditions of work; and Habit is therefore to be classed with those factors which are due to a special education of attention.

The writer then proceeds to compare Kraepelin's laws with the results of his own most recent experiments on muscular voluntary work. He finds that the "psychic inertia," a phenomenon assigned by Kraepelin to individual temperament, appears also in muscular work; and he finds many points of similarity between his own curve of Acceleration and the curve of *Antrieb* (or accentuation of Will Tension) in mental work.

The paper concludes with an enquiry into the ultimate nature of the determining psychic element in voluntary work.

The writer reviews very briefly the results obtained from the

experiments of Kraepelin, Lehmann, and Loeb; and concludes that the complex of facts thus obtained "squares well enough with the doctrine of William James, according to which the effort of attention is the essential phenomenon of the will," and that the value of the Acceleration in muscular, and that of the *Antrieb* in mental work, may therefore be regarded as the measure of the general tone of attention. MARGARET DRUMMOND.

## PSYCHOLOGY

**CONSCIOUSNESS.** (*La Notion de Conscience.*) JAMES, *Archiv. de* (240) *Psychol.*, June 1905, p. 1.

It has come at last; the philosophers have been preparing us for it for years. Berkeley has already given Matter the *coup de grâce*; and now Professor James tells us that Mind does not exist any more than Matter. What *does* exist and form the part of reality which is connoted by the word "Mind" is the susceptibility which items of experience possess of being recalled and known.

This susceptibility is explained by the fact that certain experiences may lead to one another by intervening experiences of such a nature that some play the rôle of things known, while others play the rôle of knowing subjects. These two rôles can be clearly defined without going beyond the web of experience itself, and without invoking the transcendental.

The terms subject and object, thing and idea-of-the-thing, the thing represented and its representation, imply a practical distinction which is of trivial importance, of a functional nature only, and in no way ontological as classical dualism represents it to be.

Lastly, things and ideas are not fundamentally heterogeneous, but are made of the same stuff, which cannot be defined, but which one may call, if one wishes, the stuff that experience in general is made of. W. H. B. STODDART.

**THE CURVE OF INTELLECTUAL WORK ACCORDING TO** (241) **KRAEPELIN.** (*La courbe du travail intellectuel d'après Kraepelin.*) N. VASCHIDE, *Rev. de Psychiat.*, May 1905, p. 188.

THIS paper is devoted to an exposition of the studies of intellectual fatigue carried out by Kraepelin and his pupils during recent years. Some of these studies have been published in a collected form, under the title "Die Arbeitscurve."<sup>1</sup>

<sup>1</sup> W. Engelmann, Leipzig, 1902 (*Wundt's Festschrift-Philosophische Studien*. Bd. xix.).

The paper is divided into three parts—

I. *The Curve of Intellectual Work*.—The principal method of experiment has been the performance of simple intellectual operations as rapidly as possible for definite times—such operations, for instance, as the addition of numerals composed of single figures. It is found that the curve obtained (*i.e.* that indicating the numbers counted in equal times) rises and falls in accordance with various conditions. The chief of these are—

(1) Fatigue. This causes a fall in the curve, and its influence becomes more marked the longer the work is continued.

(2) Practice. This causes a rise in the curve, and is therefore opposed to fatigue. The respective influence of these two factors is modified by various circumstances pertaining to the subject or to the conditions of the experiment.

(3) Time of day. The time of the experiment is one of these modifying circumstances, the curve tending to fall sooner when the experiment is done in the evening than when it is carried out in the morning. But this factor itself is modified by the habits of the subject, for it has been found that in persons who are accustomed to do their work in the evening the influence of fatigue may be best marked in the morning.

(4) Food. After food the capacity for work is diminished, but this diminution is not true fatigue.

(5) Stimulants. After the paralysing effects of alcohol or ether have passed off the curve rises, and continues to rise as the work is pursued.

(6) The will. The influence of fatigue is often diminished or counteracted by the influence of the will. This is often seen when the conditions of the experiment allow the subject to perceive that the curve is falling, or when the subject knows that the time of the experiment is almost completed.

Taking all these factors into account, we cannot regard the curve of work as an expression of the relationship between practice and fatigue. We can regulate the external conditions of our experiments, but there are others over which we have not the same control.

II. *The Influence of Pauses upon Mental Work*.—If one repeats, after a short interval, an exercise which has not been practised, one finds that the values obtained (beginning, end, and maximum elevation) are much superior to those first noted. This proves that the effects of fatigue disappear quickly, whilst those of practice leave durable traces. These traces may last for a very long time. A *part* of the gain from practice is quickly lost, but the remainder diminishes very slowly. Two subjects who worked at additions for an hour daily, gained in the first half hour of the second day 545 and 299 figures (in relation to the first day), while the mean

daily increase from practice during ten days for the first half hour was 145 and 150 figures. This result being different from the usual progress of the curve is due to a new factor, and this is *habit*.

Fatigue resulting from work can be compensated only by rest, and in the case of intellectual fatigue, complete compensation usually occurs only after sleep. Taking into account the gain from practice, the conclusion may be stated that after fatiguing intellectual work the same degree of production can be again arrived at only after sleep has repaired not only the fatigue due to the experiment, but also the ordinary fatigue of the day.

A rest after fatiguing work has the effect of increasing the capacity for work. The capacity for work, thus increasing, may eventually surpass its original value, owing to the result of the previous practice. The attainment of the highest capacity for work determines the point at which the surplus resulting from practice is greatest over the residual results of fatigue. If a longer rest is taken, the increasing loss of the gain from practice is accompanied by a loss in the capacity for work. There exists, therefore, a *favourable pause*, which may be approximately determined by experiment.

The effect of the pause varies according to the duration and the nature of the work. Thus Amberg has shown that a rest of fifteen minutes after working at addition for an hour augments the capacity for work, while a rest of the same duration after working for half an hour has an unfavourable effect.

III. *The Construction of the Curve of Intellectual Work.*—The various phenomena which play a part in the building up of the curve are so intimately related that it is extremely difficult to gauge their respective influences. In attempting to do so, Kraepelin studied especially the effect of pauses of very variable duration. For example, to ascertain the effect of practice he made use of experiments of five minutes' duration with rests of thirty minutes. It will be remembered that the fatigue resulting from a short experiment passes off very quickly, while the effect of the practice endures.

To ascertain the effect of fatigue he made use of experiments continued for a prolonged period without rest. Thus, if work is continued for an hour, the amount done in the fourth quarter of an hour should surpass the amount done in the second quarter by the value of half an hour's practice. The amount it falls short of this indicates the fatigue of half an hour's work.

The influence of impulsion is calculated by comparing a series of experiments without pause with the results obtained after a rest of five minutes.

The influence of habit and of will may also be calculated, but Kraepelin admits that the results are somewhat doubtful.

The curve of work itself is nearly horizontal, with variations due to the will. Practice and fatigue, acting in opposite ways, almost balance one another. Impulsion and habit result in a greater amount of work than would result from the influence of the will alone.

Kraepelin's diagram, showing the various factors entering into the composition of the curve, is reproduced. This diagram has separate lines indicating respectively the normal curve, practice, habit, excitation (impulsion), will, and mental fatigue.

W. B. DRUMMOND.

### PATHOLOGY.

**CONTRIBUTION TO THE ANATOMY OF ACUTE ANTERIOR (242) POLIOMYELITIS.** (*Beiträge zur Anatomie der Poliomyelitis anterior acuta.*) D. RUDOLF NEURATH, *Arch. aus. d. neurolog. Inst. in Wien.*, 1905, p. 297.

AFTER discussing the various views which have been held with regard to the pathology of acute anterior poliomyelitis, the author describes the case of a boy aged five who toward the end of May had some inflammation of the throat, which lasted two days. At the beginning of August the boy had a febrile attack, with loss of appetite, pain in the arms and legs. This attack lasted five days, and was attributed to rheumatism, although there was no swelling of the joints. At the end of August a similar attack occurred again, lasting five days, the boy recovering his usual health.

A second child developed scarlet fever about this time, so that the boy was sent to relations.

The boy was taken ill again in October, with the same symptoms as on the former occasion, and he lost the use of his arms and legs. On examination, a flaccid paralysis of the neck, arm, and leg muscles. The boy could move the fingers and the toes. All the deep reflexes of the lower extremity were lost.

The cranial nerves were not affected, except for a certain alteration in the articulation. There was no loss of consciousness.

Breathing was superficial and purely thoracic.

The power of swallowing was impaired.

Death occurred two days after the onset of the disease.

Examination of the spinal cord showed marked hyperæmia of the grey matter, with exudation of small round cells, extending from the sacral to the cervical region, this being especially marked in the anterior horn. The vessel walls were infiltrated, and the peri-vascular space distended with round cells. The anterior spinal artery did not appear more affected than other

vessels of the pia, but the artery of the anterior sulcus was intensely affected.

The ganglion cells of the anterior horn were diminished in number, and showed marked changes, a few normal cells being present; but all stages were present from a perfectly normal cell to complete atrophy.

The intensity of the change gradually increased from the cervical to the caudal extremity of the cord. The pathology of the condition is discussed from the standpoint of this case, and the view is taken that both the inflammatory processes and the cell alteration are due to some toxin circulating in the blood. No organisms were found in the sections examined.

A second case is described in a child who died when seventeen months old, the paralysis having existed for ten months. The changes found were chiefly of the anterior horn, and diminution and absence of cells at certain levels of the spinal cord.

The clinical symptoms were not completely in accord with the pathological findings, since there was considerable atrophy of the cells of the anterior horn in the cervical region without any corresponding clinical symptoms.

F. E. BATTEN.

#### ON THE PATHOLOGY OF WARREN-TAY SACHS AMAUROTIC

(243) IDIOCY. (Zur Pathogenese der Tay-Sachs-schen amaurotischen Idiotie.) KARL SCHAFER, *Neurolog. Centralbl.*, May 1, 1905, p. 386 *et seq.*

THE following are the changes found in the condition :—

- (1) Swelling of the whole nerve cell.
- (2) Partial swelling of the cell body with the main dendrite, so that an appearance of cystic degeneration is obtained.
- (3) In the more healthy portions of the cell body and dendrites, the neurofibrils are separated from one another, and in later stages appear as a mass of detritus.
- (4) Hypertrophy of the glia tissue is secondary to these changes.
- (5) The blood-vessels are normal, no trace of inflammation being present.

The author found changes in the fibrillary reticulum of the nerve cells. And in comparing the changes in the nerve cells as shown by the Nissl and the Bielschowsky method, it was of interest to note that even when a cell showed advanced chromolysis, the nerve fibrils of the cells might show but little change. This would seem to show that the Nissl substance of the cell was more susceptible than the nerve fibrils. This explains the fact that a nerve cell, which showed marked tigroid change,

might show no change in function. The disease affected the whole central nerve system, and no cell group escaped.

The author was unable to find any change in the optic nerve by any method of examination, and believes that the blindness is to be explained by change in the central nervous system. Marked changes were found in the cortex in the region of the calcarine fissure.

The deficiency in the medullated fibres of the cortex is marked in the frontal lobes and in the temporal lobes. The pyramids were always sclerosed.

The primary change is in the interfibrillary substance of the cell, the change in the fibrillary substance being secondary.

The wide extent of the pathological process and the primary character of this disease point to a subnormal development of the nervous system, which tends to degeneration when called into function. One of the most striking features of the disease is that neither vascular nor infectious processes play any part in its production. For the assumption of an auto-intoxication we have at present no evidence.

F. E. BATTEN.

#### **A CONTRIBUTION TO THE PATHOLOGY OF SCIATICA.**

(244) J. RAMSAY HUNT, *Amer. Med.*, April 15, 1905, p. 620.

AFTER a short summary of eleven cases of sciatica, examined post-mortem, recorded in the literature, the author describes a typical case which was treated in the Bellevue Hospital, and there died of pneumonia. At autopsy the nerve trunk was found to be distinctly enlarged and thickened. This was due to the deposit in the epineureum and epineural fat of a firm translucent substance of gelatinous consistency. There was no redness nor congestion, nor any sign of inflammation. Examined histologically, the nerve was entirely negative as far as inflammatory changes or signs of degeneration of the nerve fibres are concerned, and no changes were found in the muscles supplied by the nerve.

The author considers that this is a special type of the disease, and suggests its probable relation to gouty and rheumatic states.

COLIN K. RUSSEL.

#### **LESIONS OF THE NEURO-FIBRILS IN CERTAIN PATHO-**

(245) **LOGICAL CONDITIONS.** (*Lesions des neuro-fibrilles dans certain états pathologiques.*) MARINESCO, *Journ. de Neurol.*, juin 20, 1905, p. 221.

MARINESCO in this paper gives the results of an investigation into the changes which are met with in the neuro-fibrils of nerve cells

in various pathological conditions — myelitis, meningitis, and softenings of the brain. The reactions which the neuro-fibrils exhibit when acted on by the toxic or physical agents are seen in the form of pallor, a granular state, disintegration partial or generalised, fragmentation, degeneration partial or general, or in a thickening more or less widely spread.

These changes may commence at the centre or at the periphery of the cell, or diffusely. So also may the condition of chromatolysis, in fact the changes in the neuro-fibrils and in the chromatophile elements as a rule occur and progress together.

In those cases in which the neuro-fibrils are simply disintegrated into fine dark-coloured granules, recovery is still possible. But this disintegration may pass on to a granular degeneration, in which the fibrils are completely disorganised, and in that case recovery is impossible.

Sometimes also vacuoles are seen in the cells; these are produced by a destruction of the network of fibrils. In the condition which Marinesco has previously described under the term "achromatosis," the neuro-fibrils have always entirely disappeared.

In many cases the nucleus is displaced towards the apex, towards the base, or towards the periphery of the cell, and then it has been found that the fibrils are affected most in the regions of the cell which are farthest away from the nucleus; the area of atrophy is greatest when the nucleus is displaced to the periphery.

The fibrils of the protoplasmic processes are affected later.

The intensity of the lesions in the acute inflammations depends on many factors, among which the author mentions—the nature and the virulence of the infective agent, and the duration of its action. Then it is to be remembered that inflammation is always accompanied by vascular troubles, such as congestion, transudation, and anæmia, and it is also accompanied by fever. All these factors intervene in the production of the histological lesions of the fibrils.

R. G. Rows.

**LESIONS OF THE ENDOCELLULAR FIBRILLARY RETICULUM**  
(246) **IN THE CELLS OF THE CORD FOLLOWING TEARING**  
**OUT OF THE SCIATIC NERVE AND THE CORRES-**  
**PONDING POSTERIOR ROOTS.** (*Lesioni del reticolo fibril-*  
*lare endocellulare nelle cellule midollari per lo strappo dello*  
*sciatico e delle relative radici spinali.*) DONAGGIO and  
FRAGNITO, *Riv. Sper. di Freniatria*, June 1905.

THE authors here publish the results obtained by tearing out the sciatic nerve of rabbits. The pieces taken from the cord were



treated according to the third, fourth, and fifth methods of Donaggio.

The following were the principal results obtained :—

1. The endocellular fibrillary reticulum in the motor cells of the cord, on the same side as the lesion, showed, on the second day, the meshes compressed and more regular.

2. This condensation was very marked on the fifth day after the operation, and was associated with a swelling of the cell. With method 3, and to a less extent with method 5, the reticulum stained less definitely, while the whole cell was more coloured than usual. The fibrils of the protoplasmic process were also more faintly stained.

3. Towards the tenth day the fibrils again take the stain normally, but the reticulum is disturbed and altered in its arrangement, so that vortices and spirals are found.

4. By the fifteenth day the reticulum is less disturbed, and in some cells it has a regular form ; it is stained more deeply than usual.

5. The cell now gradually atrophies, and the reticulum slowly follows the cell in its atrophy.

6. The phases described above are characteristic of the motor cells only ; nothing similar is seen in the other elements of the grey matter.

7. In some of the other elements on the same side, and a few on the other side, the nucleus, which usually in method 3 remains unstained, after the operation showed that it had not differentiated but was strongly stained, while the reticulum presented a coloration less than normal.

8. The peripheral long fibres often failed to show this altered coloration. The resistance of the nucleus to decoloration was also seen in some of the motor cells, but to a much less extent.

9. Occasionally the fibrils seemed thickened, possibly from the fusion of several fibrils.

10. The lesions of the reticulum are more profound and more rapid than in the motor cells. This is proved by the fact that a true cellular disintegration is observed on the fifth day after the operation.

11. The lesions in the segment above are also characteristic. Only scattered cells are affected, and these are chiefly situated in the postero-external part of the anterior cornu. One of the most typical changes consists in the formation of a dense band by the peripheral long fibrils, so that it is often difficult to distinguish the separate fibrils. The reticulum itself is remarkably clear. In other cells there is a rarefaction of the reticulum in the middle of the cell, and a condensation at the periphery (homogeneisation of the fibrillary reticulum and of the long fibrils).

12. Comparing the results obtained by using Donaggio's method with those obtained by the method of Ramon y Cajal, it is found that while Cajal, after five days, is unable to see any trace of fibrils in the cells of the anterior cornua corresponding to the roots torn out, Donaggio, with methods 3 and 5, shows that the reticulum is well preserved and even more dense.

13. It is doubtful whether the grave and rapid destructive lesions described by Marinesco, after tearing out the hypoglossal nerve, may not be due, at least in part, to the technique used.

14. After tearing out the sciatic nerve and the corresponding roots, the resistance of the fibrillary reticulum of the cells of the anterior horn appears to be greater than in those of the posterior horn.

R. G. Rows.

**PERSISTENCE OF AN INTACT BUNDLE IN THE OPTIC TRACTS AFTER COMPLETE ATROPHY OF THE OPTIC NERVES: THE "RESIDUARY BUNDLE OF THE TRACT"—THE BASAL OPTIC GANGLION AND ITS CONNECTIONS.** (Persistance d'un faisceau intact dans les bandelettes optiques après atrophie complète des nerfs: Le "faisceau résiduaire de la bandelette"—Le ganglion optique basal et ses connexions.) PIERRE MARIE and ANDRÉ LÉRI, *Rev. Neurolog.*, May 30, 1905, p. 493.

THE authors made sections of the optic tracts in nine cases of tabes or combined sclerosis with blindness; they found in each tract a small bundle of fibres always in the same situation. But, in most of the cases, no fibres were found in the optic nerves; this bundle was quite independent of the visual fibres, whose cells of origin are in the retina. The writers believe that in every case of blindness due to tabes or combined sclerosis a small bundle of intact fibres remains in the tract. They give the following résumé of their paper:—

1. Following complete tabetic atrophy of the optic nerves, a bundle remains in the tracts which occupies the external part and in consequence is quite distinct from the commissure of Gudden: they call it the residuary bundle of the tract (*faisceau résiduaire de la bandelette*).

2. Posteriorly, this bundle ends beyond the termination of Meynert's commissure, in the fibres which border the inferior part of the lenticular nucleus.

3. Anteriorly, it reaches the basal optic ganglion of Meynert (*noyau susoptique de Lenhossek*), and evidently is related to it.

4. The true basal optic ganglion, situated above and external to the origin of the tracts, should be distinguished from the

nuclei of the tuber cinereum situated below and internal to them.

5. The basal optic ganglion is the rendezvous of a large number of fibres which are: posteriorly, the "residuary bundle of the tract"; anteriorly, a sort of fan of fibres which covers the chiasma, and which includes: (1) externally, a small inconstant bundle, which deviates towards the external margin of the superior surface of the chiasma, and which appears to be directly continuous, in part at least, with a part of the residuary bundle of the tract; it may be called the anterior residuary bundle (*faisceau résiduaire antérieur*), or the residuary bundle of the chiasma (*faisceau résiduaire du chiasma*); (2) internally, a tract of scattered fibres which border the ventricular ependyma, sub-ventricular fibres; generally none of these fibres go beyond the anterior part of the chiasma. Internally, some fibres of Meynert's commissure, probably after crossing. Above, some fibres go to the medullary lamina of the thalamus, and likely to the central grey substance.

The basal optic ganglion, which is an important cell mass, is very probably a "relay" for a great part of these fibres, but this is not proved.

6. These fibres and bundles may often be recognised, when one knows their situation, in sections of normal optic paths.

C. H. USHER.

#### THE ACTION OF RADIUM RAYS ON THE CENTRAL NERVOUS

(248) **SYSTEM.** (*Über die Wirkungen der Radiumbestrahlung auf das Centralnervensystem.*) OBERSTEINER, *Arb. aus d. neurol. Inst. in Wien*, 1905, p. 86.

It was first shown by Danysz that the application of a salt of radium, enclosed in a glass tube, to the head or spinal column of an animal produced paresis, ataxia, convulsions, and even death; that older animals were less susceptible, probably owing to their thicker bones; and that the intensity of the symptoms and rapidity of their onset were dependent as much on the purity of the radium as on the length of its application. Danysz further found that on post-mortem there were hæmorrhagic lesions of the central nervous system, but that the changes in the nerve elements themselves were not constant.

Other observers have found hæmorrhagic lesions, and Grünbaum a hæmolytic action on the blood.

Obersteiner employed radium bromide in capsules, one containing 10 m.g. and another 50 m.g. White mice were used for the experiment, and each was enclosed in a metal box which

allowed little movement of the animal, and had an aperture above the head for application of the capsule. After 24, 48, 72, or 96 hours the mouse was removed, placed in a glass-walled box and watched. Thirty-six of these experiments are recorded; the mice were young and old, and in some the 10 m.g. capsule was used, in others the 50 m.g. The results varied considerably; some showed paresis, paraplegia, paralysis of the sphincters, and died more or less rapidly; others had no prominent nervous symptoms, but died a few days after the exposure; while yet others survived an exposure of 72 to 96 hours. In the latter case there seems to have been some doubt about the radio-activity of the 10 m.g. specimen. There were few controls, and of these two died after enclosure in the box, although radium was not employed. Obersteiner believes that this was due to some after action of the radium, for it only occurred when the animals were put into the box immediately after it had been used.

Post-mortem examination showed in nearly all cases marked hyperæmia of the brain and spinal cord with hæmorrhages and small celled infiltration of various parts. Hæmorrhages were most numerous and large in the spinal cord, and were sufficient to account for the paraplegia.

The nerve cells of the brain and spinal cord showed a certain amount of chromatolysis and the appearance of endocellular channels in them. In one case the nuclei of the anterior cornua cells of the spinal cord were much altered, stained deeply with methylene blue, and presented a star-shaped or mulberry appearance. Fatty changes were found in the capsules of the cells of the spinal ganglia and in the endothelial cells of the capillaries.

Obersteiner concludes that radium rays have no elective action on the central nervous system unless it be their production of fatty degeneration of the vascular endothelium; the widespread changes that occur are the results of a general disturbance of the circulation and metabolism.

PERCY T. HERRING.

**THE INFLUENCE OF RADIUM RAYS ON MUSCLES AND  
(249) PERIPHERAL NERVES.** (Ueber den Einfluss der Radium-  
bestrahlung auf Muskeln und periphere Nerven.) EIKITSCHI  
OKADA, *Arb. aus d. neurolog. Inst. in Wien*, 1905, p. 105.

THE work was done in Vienna, and is a supplement to that of Obersteiner. The 10 m.g. capsule of radium bromide was employed, and its action on the muscles and peripheral nerves of mice and rabbits investigated; some of the mice had previously served for Obersteiner's experiments. The muscles of the back were stained by Marchi's method, and showed some fatty change, especially in

the fibres of the surface; the nerves were unaffected. In four young rabbits the capsule of radium was fixed directly over the sciatic nerve, the limb having been first shaved; application was made twice daily for from two to seventeen days.

Beyond the formation of ulcers in the skin and dermatitis, no change could be detected, the sciatic nerve and muscles were unaffected; even in the inflamed skin the nerve fibres had a normal appearance.

PERCY T. HERRING.

**THE ANATOMY OF THE CENTRAL NERVOUS SYSTEM IN  
(250) A TERATOLOGICAL SPECIMEN. (Zur Anatomie der Zentralnervensystem einer Doppelmissbildung bei der Ziege.)**  
GERTRUD BIER, *Arb. aus d. neurol. Inst. in Wien*, 1905, p. 282.

THE specimen of mal-developed goat described here belonged to the class "dipygus tribrachius," that is, there were three upper limbs, and the trunk below the umbilicus was doubled.

The examination of the available parts of the central nervous system showed, within the skull, duplication of the hypophysis cerebri and the existence of two supernumerary mesial emerging nerve-trunks, which, from their position, were an additional sixth and tenth cranial nerve respectively.

The spinal cord was apparently normal down to the fifth cervical segment, where a mesial, or third, fifth cervical nerve was found emerging from the spinal canal. Below this level, the spinal cord bifurcated into two apparently symmetrical and complete cords. Tracing the process of dichotomy of the cord downwards, the first apparent change was a duplication of the anterior median fissure, which passed from the commissure to the surface of the cord in two diverging limbs.

These two limbs lower down diverged at their root, and the division was carried back to the posterior fissure, so that there was the appearance of a Y-shaped fissure.

The central area between the limbs of the Y increased in size, and lower down became resolved into two complete anterior and posterior cornua, surrounded by white matter. A depression appeared on the surface of the cord, and divided the cord lower down into two almost symmetrical and normal complete cords.

This account is interesting; but as the specimen was not in very good condition for microscopical examination, several interesting and instructive points could not be made out.

The author discusses some points in the ætiology of the condition.

DAVID WATERSTON.

**CLINICAL NEUROLOGY.**

**A CONTRIBUTION TO THE STUDY OF THE RELATION OF**  
**(251) GENERAL PARALYSIS AND TABES DORSALIS. HENRY**  
A. COTTON, *Am. Journ. of Insanity*, Vol. lxi., No. 4, April 1905,  
p. 581.

DR COTTON gives an interesting sketch of the gradual recognition of the relationship between tabes and general paralysis. Westphal appears to have been the first observer to describe a case of general paralysis and co-existing tabes. Raymond, Nageotte, Fournier, Gaupp, Schaffer, and Mott have done much to establish the relationship. The author is a firm believer in the association between syphilis and general paralysis. In the Worcester Insane Hospital, the percentage frequency of syphilis in general paralysis rose from 25 per cent. in 1896 to 75 per cent. in 1901, the only reasonable explanation of this increase being the special attention directed to the point in the examination of cases.

Twelve cases of tabo-paresis are described clinically, and the pathological changes observed in nine of these cases are detailed. Finally, from a study of the literature and of the above cases the conclusions arrived at are:—

1. That clinically tabes and general paralysis present many analogies in etiology, symptomatology, and course.
2. That their occurrence in the same individual is more than a coincidence.
3. That in those cases of tabo-paralysis the symptoms presented are identical with the symptoms of general paralysis and tabes when seen apart, only differing in degree, according to the extent of the anatomical lesion.
4. That the clinical symptoms of tabo-paralysis have the same anatomical basis as in the separate diseases.
5. That anatomically the affection of the posterior columns of the cord, as seen in tabo-paralysis, does not differ from the picture presented in pure tabes. The same systems are affected and the segmental character of the process is the same, also that the process in the cortex is identical with that of general paralysis.
6. While the above facts show the intimate relation between general paralysis and tabes dorsalis, the unsettled status of their pathogenesis at present prevents their identity being absolutely established on an anatomical basis.

EDWIN BRAMWELL.

**TABES DORSALIS AND MENTAL DISEASE.** (*Tabes dorsalis* (252) *und Psychose.*) M. BORNSTEIN (of Warsaw), *Monatsschr. f. Psych. u. Neur.*, Bd. 17, *Ergänzungsheft*, 1905.

SOME authors regard tabes dorsalis and dementia paralytica as essentially the same disease, and consider all mental disorders in tabes as either of paralytic nature, or as casual complications of tabes, and easily recognised as such. Bornstein calls attention to the difficulty of diagnosing such complications of tabes with a psychosis from general paralysis in view of the protean forms of the latter. General paralysis may be preceded by other psychoses; the early stage of general paralysis may closely simulate other conditions, *e.g.* paranoia, mania. The somatic symptoms, which enable us to diagnose general paralysis from other psychoses are of little use when the other psychoses are associated with tabes: a tabetic with a catatonic stupor is difficult to diagnose from a case of tabo-paralysis.

Even the nature of the paralytic deterioration may be insufficient to establish the diagnosis until its progressive course makes the condition clear.

Notwithstanding the difficulty of eliminating general paralysis there has been established the co-existence of other psychoses with tabes. Taking the cases of Meyer (56) and Schultze (2), *i.e.* 58 cases of non-paralytic psychoses accompanying tabes, 22 were chronic hallucinatory paranoia (using the term in a wide sense), 15 were depressions (melancholia, hypochondria). These cases showed no difference in their course from other cases of the same psychoses, not associated with tabes.

The author records the case of a male tabetic, aged 33, whose mental disorder came on three months after the onset of the first symptoms of tabes (lightning pains), and two months after, patient's eyesight began to fail. His mental disorder consisted of a succession of attacks which were characterised by visual, auditory, and tactile hallucinations, and by disorientation.

In the psychoses complicating tabes, hallucinations of various senses are frequent, and also depression partly based on physical and mental suffering. Visual hallucinations predominate, and were in the above cases always associated with atrophy of the optic nerve. The frequency and variety of hallucinations may be perhaps referred to a hyperæsthesia of the sensorium analogous to the more peripheral hyperæsthesia causing various pains and paræsthesias. Hallucinations in tabes may be part of a chronic hallucinatory paranoia, or of an acute hallucinatory confusion; but one meets also transitory periods of excitement and hallucinations without the development of a definite psychosis.

Bornstein considers the question of the characteristics of the cases of tabes which develop mental symptoms of a non-paralytic nature. Female tabetics develop psychoses much more frequently than male tabetics. Cases of tabes with non-paralytic psychoses have more frequently implication of the optic nerve and the motor nerves of the eye than tabetics without mental complications, or than tabetics with general paralysis. Tabetics with psychoses show a higher percentage of bad heredity than simple cases of tabes.

The connection of the tabetic process with the psychosis is not clear; no definite pathogenetic connection between the tabetic process and the psychosis has been established, but the malnutrition, ataxia, and sensory disorders, which furnish food for false elaboration, give a special colouring to the psychosis.

In conclusion, the author insists that there are not sufficient grounds for the assumption of a special tabetic psychosis, but that the mental disturbances in tabes cannot be looked upon as casual associations.

Tabetics of good mental constitution may, on the ground of sensory disorders, develop hallucinations without a psychosis, while in the case of patients with a predisposition to mental disease the same conditions may lead to the development of a psychosis.

C. MACFIE CAMPBELL.

**TUMOURS OF THE CEREBELLUM.** (1) The Diagnosis of Tumours (253) of the Cerebellum, especially with reference to their Surgical Removal. CHARLES K. MILLS. (2) Remarks upon the Surgical Aspects of Tumours of the Cerebellum. CHARLES H. FRAZIER. (3) Cases illustrating the Papers of Dr Mills and Dr Frazier. (4) The Ocular Symptoms of Cerebellar Tumours. GEORGE E. DE SCHWEINITZ. (5) The Pathology of Cerebellar Tumours. T. H. WEISENBERG. (6) The Functions of the Cerebellum. EDWARD LODHOLZ.

UNDER the title of "Tumours of the Cerebellum," the authors have published in book form several papers which have appeared in the *New York Medical Journal* and *Philadelphia Medical Journal* of this year. The first article is contributed by Dr Charles K. Mills, and in it he discusses the diagnosis of tumours of the cerebellum, with special reference to their surgical removal. This paper is of interest, but one regrets that the author has not entered more fully into some of the points which he mentions, such as cerebellar vertigo. Dr Mills considers that asthenia or muscular



weakness, especially of the trunk muscles, is a true cerebellar symptom; but he makes no reference to hemiparesis except as a symptom of ponto-bulbar pressure. He does not regard cerebellar nystagmus or hemiasynergia as reliable signs. In discussing the gait of cerebellar cases, he holds that the tendency to fall is towards the side of the lesion, and he lays stress on this as a localising sign. He states that cranial nerve symptoms may be referable to any of the cranial nerves from the third to the twelfth, but that the sixth, seventh, eighth, and fifth are the most frequently affected. It is evident, however, that the author includes cases in which the growth invades or exerts pressure on parts outside the cerebellum, as he states later that the cranial nerve symptoms may be used not only in focal diagnosis, but also in prognosis, as indicating whether the growth is confined to the cerebellum or not. The last portion of the paper is devoted to the description of tumours of the cerebello-pontine angle, and special reference is made to the diagnosis of fibromata of the eighth, seventh, and fifth cranial nerves.

Dr Frazier writes about the surgical aspect of tumours of the cerebellum. He describes the technique he employs in the various operations which may have to be performed in this region. It may be noted that he uses a chisel in preference to the trephine in making the initial opening into the skull. The question as to a one or a two stage operation he decides on general principles, and on the special circumstances of each case. The concluding step in the first stage is the opening of the dura by turning down a flap. This procedure is almost invariably followed by protrusion of the cerebellar tissue and consequent laceration, so that it is open to question whether it should be included in the first stage or postponed until the second. In discussing the removal of tumours from the cerebello-pontine angle in cases where the cerebellum cannot be retracted sufficiently, he mentions two methods which can be resorted to: (1) Excision of a portion of the cerebellum. (2) Puncture of the ventricles. This latter procedure he very rightly condemns. The results of lumbar puncture as a palliative measure scarcely warrant its employment. Dr Mills' and Dr Frazier's papers are illustrated by clinical details of six cases. Dr G. E. de Schweinitz has written an admirable article on the ocular symptoms of cerebellar tumours. His observations will be fully endorsed by all who have had the opportunity of studying such cases. The pathology of cerebellar tumours is discussed by Dr Weisenburg in a paper which contains much useful information. Dr Edward Lodholz contributes the final paper, on the functions of the cerebellum. He reviews in a clear and concise manner the investigations which have been made on that subject.

T. GRAINGER STEWART.

**THE COURSE OF OPTIC NEURITIS AND ITS SUBSIDENCE  
(254) AFTER OPERATION IN CASES OF CEREBRAL TUMOUR.**

LESLIE PATON. Abstract of a Paper read before the Ophthalmological Society, June 8, 1905.

THE paper is the result of the analysis of cases operated on at the National Hospital, Queen Square, Bloomsbury, during the years 1903 and 1904. Out of 105 cases of cerebral tumour, 20 showed no signs of optic neuritis. Of the remaining 85, 38 were not operated on; and out of the 47 cases operated on, 17 died shortly after operation, or were never in a condition which allowed a proper examination to be made. This paper is based on the examination of the course of the optic neuritis in the remaining 30 cases.

**VISUAL ACUITY.**

It was found that the optic neuritis affected the visual acuity in various ways in different cases. These fell more or less accurately into four classes.

1. The visual acuity remained practically unaltered during the whole course of the optic neuritis, or was only very slightly diminished. This class included 8 cases.

2. The visual acuity was markedly diminished at the time of operation. In some cases it was reduced to bare perception of light. Recovery of vision took place after operation, and the recovery was in some cases almost complete, so that the visual acuity was normal. This occurred in 11 cases.

3. Sub-divided into two classes.

(a) Cases practically blind at operation, and regaining no vision (6 cases).

(b) Cases in which failure of vision took place subsequent to, and in spite of operation (2 cases), a total of 8 cases out of 30, in which operation did not save sight.

4. Three cases in which one eye retained good vision, while the other became blind, or nearly so.

Out of the 30 cases useful vision was saved in 22. In none of the cases in which the sight was lost was the optic neuritis more severe than it was in some of the cases in which perfectly good vision was retained.

**TRANSIENT ATTACKS OF DIMNESS OF VISION.**

This subjective symptom occurs in many cases of cerebral tumour with optic neuritis. In the present series it occurred in 6 cases of cerebellar tumour, 2 frontal, 1 parietal, and 1 case of pontine tumour. In another series of 38 cases examined by the author it occurred

4 times, and these were all cases of cerebellar tumour. The explanations offered for this phenomenon are mostly unsatisfactory. It has been for the most part assumed that it is due to a temporary increase in intra-cranial pressure, causing a strangulation of the optic nerve either in the foramen or behind the disc. Its frequent occurrence in cerebellar cases, the short duration of the attack (usually not longer than 3 or 4 minutes), and the fact that it is usually accompanied by an attack of giddiness or by increased headache, point to its being due in some way to intra-cranial disturbance. This, however, may be vascular, and due to an interference with the blood supply of the occipital lobes. Another possible explanation is that in cerebellar cases and sub-tentorial cases there is a greater tendency to a rise in the intra-ventricular as distinct from the intra-cranial pressure. This gives rise to great distension of the infundibulum in some cases, and the temporary loss of vision may be caused by the pressure of the distended floor of the third ventricle on the chiasma.

#### RELATIONSHIP BETWEEN VISUAL LOSS AND POSITION OF TUMOUR.

There were 5 cases of frontal tumour in the series. Of these, 3 belonged to Class 1, 1 to Class 2, and 1 to Class 4. In 11 cases of parietal tumour, 4 belonged to Class 1, 3 to Class 2, 1 to Class 4, and 3 to Class 3—i.e. where sight was lost. Out of 11 cerebellar cases, 1 came into Class 1, 6 to Class 2, and 4 to Class 3. The only pontine case belonged to Class 3.

#### THE OPHTHALMOSCOPIC APPEARANCES.

The optic neuritis commences by an increase in the colour of the disc with blurring of the upper and lower edges and slight turgescence of the veins. The blurring spreads along the inner margin and lastly to the outer margin; the physiological cup becomes filled in and obscured; the veins become more turgid; the smaller vessels on the disc, usually invisible, dilate, and a condition of steep-edged swelling of a somewhat striate and juicy appearance characterises the completion of the first stage of optic neuritis. Meantime hæmorrhages, first of all small and linear and confined to the neighbourhood of the disc, make their appearance; later these become larger and more scattered.

The average amount of disc swelling in cases of frontal and parietal tumours was from 3 to 5 dioptries, and in cerebellar cases from 5 to 7 dioptries. As the neuritis proceeds it loses its fresh juicy appearance and becomes more opaque. Patches of white exudate appear both on the disc and in the neighbouring retina. In some cases the whole disc takes on the appearance of opaque structureless glass.

## CHANGES IN MACULAR REGION.

Macular changes appear in only a small proportion of cases in this present series. In 1 frontal tumour, in 1 parietal tumour, and in 3 cases of cerebellar tumour. The macular change is of a fairly definite type. It commences by a layer of oedema, which forms a parabolic figure with its apex just inside the fovea and its base spreading out towards the disc. In its early stages this has a dirty greyish appearance. Later on the material forming it seems to gather into whitish bright dots which, towards the disc, are arranged in a series of seven or eight horizontal parallel lines. These lie in the superficial layers of the retina. Occasionally a more extensive oedema of the retina manifests itself, and when this occurs numerous close lines can be seen radiating out from the fovea as a centre. The occurrence of the first mentioned macular changes is not necessarily associated with any serious interference with visual acuity: another point in favour of the superficial position of these changes, since the percipient layers of the retina do not seem to be affected. At the time of operation intra-dural tension was definitely raised in all cases.

## THE COURSE OF THE SUBSIDENCE AFTER OPERATION.

The subsidence of the swelling was fairly steady in all the cases, usually taking about two months. In some of the cases, especially when the tumour was cystic, it was much more rapid. In the cases which went on to blindness the disc assumed an opaque papery white appearance as the swelling subsided, and in these cases the arteries became markedly diminished in size. In the other cases where sight was retained the disc, two months after operation, had usually a muddy greyish pink appearance with a dirty grey centre. As time passed this disappeared, and in cases seen at intervals, varying between eight months and two years from the time of operation, it was practically impossible to tell from the ophthalmoscopic appearances that there ever had been an attack of optic neuritis. In one case where there had been marked macular changes during the attack the vision had returned to  $\frac{5}{6}$  in each eye, and all that remained of an extreme degree of macular change was a slight disturbance of pigmentation at the macula of one eye and a similar macular disturbance in the other eye, with a small cluster of bright white dots above and to the inner side.

AUTHOR'S ABSTRACT.

**A CASE OF EXTREME MICROCEPHALY, WITH APE-LIKE (255) MOVEMENTS.** A. ERNEST JONES, *Brit. Journ. of Children's Dis.*, May 1905.

THE patient, a girl, aged 7 years, began to walk at the age of 4, but had never talked. Her mental condition was better than might have been expected from the size of her head, which measured only 13 inches in circumference. An interesting feature of the case was the lightning-like rapidity of her voluntary movements, which "reminded one irresistibly of the habitual movements of the smaller monkeys."

EDWIN BRAMWELL.

**TETANY OF PARATHYROID ORIGIN.** (*Tétanie d'origine parathyroïdienne.*) MARINESCO, *La Semaine Médicale*, 21 juin 1905, p. 289.

THE association of tetany with certain symptoms of exophthalmic goitre in the patient whose case is described leads to a consideration of the important part which may be played by another small glandular organ, the parathyroid, in the production of nervous disease. The importance of the parathyroid glands was first recognised in 1880 by a Swedish author, Sandström, but his work passed unnoticed until Gley in 1891 again called attention to the presence of these bodies. This observer was the first to show that rabbits, which did not exhibit any serious symptoms on simple thyroidectomy, developed attacks of tetany if the parathyroids were also removed, these glands differing from the thyroid in structure, development, and function. Brissaud has for a considerable time attempted to establish a distinction between myxœdema of thyroid and of parathyroid origin.

CASE. Girl aged 18. No facts of special importance in personal or family history. Illness began eight months previously by a feeling of oppression in the chest, and on one occasion also a feeling of suffocation and constriction in the throat; enlargement of the neck began a month later, and six months ago the first attack of tetany, affecting the upper limbs, supervened. Attacks recurred almost daily, each lasting from a quarter to twelve hours, and two months later they became more extensive and severe in character. The patient rapidly lost flesh, and obtained no benefit from thyroid treatment. The enlargement of the thyroid gland was more marked on the right side, fine tremor and tachycardia were present, but there was no exophthalmos. All the symptoms usually considered essential to the diagnosis of true tetany were found to be

present, *e.g.* the increased mechanical neuro-muscular excitability (Chvostek), recrudescence of tetany on percussing over the nerves distributed to the areas affected by paræsthesia (Hoffmann), and also on pressing over the nerve trunks of the arm or the vessels accompanying them (Trousseau); the increased electric excitability of the motor nerves to the galvanic stream, and an alteration in the usual order of the responses so obtained (Erb).

The author then gives a résumé of the different varieties of tetany as follows:—

1. Epidemic variety, found specially among workmen.
2. Occurring as a complication of gastro-intestinal diseases.
3. In infectious disease.
4. In toxic conditions.
5. In the female during pregnancy, parturition, and puerperium.
6. Following removal or in cases of absence of the thyroid gland.
7. As a complication of certain nervous diseases.

Pregnancy is normally accompanied by an increase in the size of the thyroid gland, and an absence of this hypertrophy may lead to very serious symptoms, such as eclampsia, renal trouble, attacks of tetany, etc., and these may improve under thyroid treatment. Pregnancy occurring in a cat from which the thyroid had been previously removed was complicated by frequent attacks of general convulsions unaffected by treatment with thyroid extract, and Vassale found this condition to be due to removal of the parathyroid along with the thyroid gland, and he produced tetanoid attacks in a pregnant bitch by partial removal of the parathyroids. Some authors assert that there is some specific connection between the parathyroids and milk secretion, and Vassale has recently successfully used the extract of these glands in the treatment of eclampsia, a condition which he regards as being due to parathyroid and not the thyroid insufficiency. Marinesco believes that the tetanoid seizures in his patient were due to perversion of parathyroid function, and thinks it highly probable that the other varieties of tetany, and perhaps also of epilepsy and eclampsia, may be traced to a similar origin. His patient has improved under parathyroid treatment, but he cannot positively assert that this has not been due to the other medical treatment employed in the case.

W. E. CARNEGIE DICKSON.

**THE HABIT NEUROSES.** (*Les phénomènes morbides d'habitude.*)  
(257) J. CROCQ, *Journ. de Neurol.*, April 20, 1905.

HABIT is one of the most common phenomena of our nervous system—every act, impression, and thought once produced will

be reproduced with greater facility the more often it traverses the interneural pathways, always tending to become more automatic, until consciousness intervenes very little or not at all. Habit so common in physiological, is still more common in pathological states. Brissaud has described habitual pains, coming on at fixed times, without apparent external influence. Meige and Feindel have declared the Tics to be due to an abnormal motor habit. The author goes further, and states all neuroses are matters of habit; the pains, obsessions, phobias, all reproduce themselves more easily the more often repeated. He reports two cases of Insomnia, in which after the causes were removed, still the patients could not sleep; both were cured by the administration of minimum doses of a hypnotic for from two to four days.

Our nervous system is an automatic machine, the functioning of which, once disturbed, requires to be regulated again in order to regain its normal equilibrium.

Three cases with hysterical attacks, caused originally by some nervous shock, and repeated at definite intervals thereafter without apparent external cause.

He describes also four cases of "Epilepsy," in all of which there were definite etiological factors, acting on a neuropathic diathesis. The attacks were repeated at regular intervals weekly or bi-weekly and usually at about the same time of day. All these cases were cured by a few administrations of a sedative before the expected attacks.

COLIN K. RUSSEL.

**BEER DELIRIUM. COMMUNICATION ON TWO CASES OF**  
(258) **HALLUCINATORY INSANITY CAUSED BY THE EX-**  
**CESSIVE USE OF BEER ALONE.** (Das Bierdelirium.  
Mittheilung zweier ausschliesslich durch Biermissbrauch  
verursachter Fälle von hallucinatorischem Wahnsinn.) Dr  
HANS GADDEN (of Munich), *Archiv f. Psychiat. u. Nervenkrankh.*,  
Bd. 40, H. 1, p. 151.

IN text-books of psychiatry and monographs on alcoholism it is generally assumed that, excepting conditions of actual intoxication, the various forms of mental disturbances associated with the abuse of alcohol result not from beer alone, but from the taking of spirits or other stronger alcoholic drink.

The writer's experience in the psychiatric clinic in Munich does not favour this view of the relative harmlessness of beer, the continued use of which, when combined with only a small quantity of "schnaps," can elicit to the full the mental phenomenon of chronic alcoholism.

Two cases are reported in which there was excessive indulgence in beer alone. Both were male subjects, the one 33, the other 39 years of age. The former had a family history of alcoholism and insanity, the latter of insanity. Both cases in their development and progress presented a clinical picture markedly characteristic of alcoholic hallucinatory insanity, and the correctness of this view was further borne out by the presence at the onset of some features of delirium (restlessness, fear, tremor, sweating).

The first patient saw little devils springing over his bed, and the second combined (in manner of a delirium case) reproachful voices into a plot originated by persons he had seen some days previously. Neither case came afterwards to have systematised fixed delusions, or to disturbances of consciousness of a deeper nature.

The duration was long, nearly two years in the first case; and the second patient had not recovered after one and a half years. Before beer drinking can produce the same mental effects as spirit drinking, a much longer period is requisite—eight litres beer corresponding, as regards amount of alcohol, to about a half litre "schnaps." The majority of those persons who limit their excesses to beer do not live long enough to develop an attack of delirium, but succumb to heart disease, tuberculosis, or nephritis, or else with the advent of these diseases they diminish or give up their drink.

As far as these two cases may justify generalisations, it would seem that besides the long duration of mental symptoms, the frightsome form of the sensory hallucination is characteristic.

A. HILL BUCHAN.

**A CASE OF SENSORY ALLOCHEIRIA.** (A propos d'un cas (259) d'allochirie sensorielle.) SABRAZÈS and BOUSQUET, *Rev. Neurolog.*, June 15, 1905, p. 573.

THE patient was a woman 48 years old, who had commenced at the age of 10 to show signs of weakness in the legs, with a staggering gait. At the age of 15 she had become quite paralytic, incapable of standing, and her left arm had begun to be feeble, amounting almost to uselessness. By the age of 20, some improvement in the right leg left her more or less hemiplegic on the left side. Five years ago the sight of her left eye gradually failed. In 1904 the condition was briefly one of left hemiparesis, with paralysis of the left lower facial group; uselessness and maladroitness of movement of the left arm, with pronounced intention tremor in the same extremity; feebleness of the left lower limb, with considerable inco-ordination accompanying any complex movement of the same; tendency to deviate or fall to the left when walking;



inclination of the head to the same side; slight spinal curvature with the concavity to the left; exaggeration of all the left tendon reflexes; flexor responses on stimulation of either sole; in the right arm a minor degree of volitional tremor, and in the right leg some general diminution of power. Not less interesting were the sensory phenomena. Thus there existed, on the first day of examination, abolition, on slight stimulation, of all forms of cutaneous sensation on the left side; stronger stimuli were perceived, but immediately localised on the right side, with a varying degree of inaccuracy. A fortnight later the anæsthesia had disappeared, and slight stimuli were at once appreciated as if on corresponding areas of the right side. The sense of vibration elicited by the application of a tuning-fork to the left side was similarly located on the opposite side. Examination with a faradic current revealed the same transference of the area stimulated to the symmetrically placed area of the right side. A powerful current, too strong to be tolerated on any part of the right side, was felt feebly, and localised accurately, on the left side. A month later, the various forms of cutaneous sensibility were abolished on the left side only as far up as the knee, and no allocheiria was elicited. A week later, this latter phenomenon had reappeared, to disappear again in the course of three days. No further examination was made for two months, at the end of which time new variations were obtained, allocheiria being definitely present in the left leg.

The diagnosis of disseminated sclerosis of a cerebello-spastic (clinical) type was made, with predominance of the lesions on one side. Unmistakable organic symptoms justified this opinion, but the authors consider that the other phenomena detailed above were of a hysterical nature. The hemianæsthesia and allocheiria, varying as they did with varying methods and times of examination, are inexplicable by any other pathogenic mechanism. If daily observations were made, the allocheiria tended to diminish and disappear; were they omitted for any length of time, the condition recurred. Prolonged repose seemed to be accompanied with some blunting of conscious appreciation of the left side of the body.

S. A. K. WILSON.

**CONTRIBUTION TO THE STUDY AND INTERPRETATION OF**  
 (260) **PALLÆSTHESIA.** (Contributo allo studio ed all' interpretazione della pallestesia.) FORLI and BARROVECCHIO, *Annal. dell' Istitut. Psichiat. dell. R. Univers. di Roma*, Vol. iii., Fasc. ii., 1904, p. 191.

IN this long and elaborate paper the whole question of the sensation of vibration is discussed in an exhaustive manner and from various

points of view ; existing hypotheses are criticised and additional clinical information is provided which is not without value. It is satisfactory to be able to note the adoption of the non-committal term *pallæsthesia*, with the corresponding *hypo-pallæsthesia* and *pallanæsthesia*, in preference to any such phrases as bone-sensation, osseous sensibility, etc. Very complete references to preceding work lead to the statement of several questions still vexed: Is the stimulus from the vibrating tuning-fork purely local (Egger), or transmitted to a distance (Minor)? Is the sensation to be attributed to stimulation of the skin (Treitel), or of periosteum and bone (Egger), or of the deeper tissues (Rydel and Seiffert), or of all the tissues (Bechterew), or of nerve trunks only (Noischewsky)?

Much existing confusion might be avoided were a standard instrument and a standard method of estimating the sensation adopted, and it is again satisfactory to observe that the authors themselves used exclusively, and recommend the use of, the tuning-fork of Rydel and Seiffert, fitted with Gradenigo's ingenious device, at once for varying the number of vibrations and for estimating the duration of the sensation.

The figures given by the two German observers for normal individuals are carefully compared with those obtained from the examination of ten healthy persons, and found not to differ appreciably in any essential particular. There is bound to be some variation within physiological limits, and it is better not to fix invariable figures for the different points of the body, even although it may thus become more difficult to define what is pathological.

To determine the latter, sixteen patients were examined, nine being cases of *tabes dorsalis*, one *tabo-paresis*, one cervical *tabes* complicated with *paranoia*, one combined *sclerosis*, two *hysteria*, one a case of *Dercum's disease* (*adiposis dolorosa*). In addition, the results recorded by former observers were collected, making a total of 94 cases, divisible into four groups.

In the first group, *pallæsthesia* and cutaneous sensation coincided accurately (20 cases).

In the second, the alteration in *pallæsthesia* resembled more the change in tactile sensibility than that in the pain and thermal senses (19 cases).

In the third, the change in *pallæsthesia* corresponded more to the alteration in the pain and thermal senses than to that of tactile sensibility (6 cases).

In the fourth, all four forms were altered in ways essentially differing (49 cases).

In a sense, then, *pallæsthesia* corresponds more to tactile than to the other forms of cutaneous sensibility, though it itself is a

special form and may present pathological alterations and variations of its own.

An interesting corroboration was furnished by the examination of three patients anaesthetised specially by Bier's method of rachicocainisation, in whom there were complete analgesia and therm-anaesthesia below a certain level, with complete conservation of pallæsthesia over the areas affected, and of tactile sensibility as well.

These facts perhaps suggest that the former may be after all only a modality of the latter (Goldscheider). But physiological and pathological evidence tends to negative this conclusion. The tongue possesses an exquisite tactile sense, yet it appreciates poorly (relatively) vibratory stimuli. The areas of pallanæsthesia and of ordinary anaesthesia do not by any means correspond in many cases. In some patients presenting the Brown-Séquard syndrome, whom Egger examined, the vibrations of the tuning-fork were perceived on the side on which there was sensory loss, but not on the paralysed side.

The authors are quite convinced, from Minor's researches and from their own, that there cannot but be a transmission, a diffusion, of the vibratory stimulus from the point of application to neighbouring tissues, both superficial and deep; they think, then, that the sense of vibration is a complex of sensations from skin down to underlying bone.

S. A. K. WILSON.

#### **INTERMITTENT CLAUDICATION AND ALLIED SYNDROMES (261) DUE TO ANGIO-SCLEROSIS OF THE EXTREMITIES.**

J. RAMSAY HUNT, *Med. Record*, May 27, 1905.

SINCE Charcot's graphic description of this complex of symptoms in 1858, many monographs on the subject have appeared in France, and later in Germany, where in 1898 Erb, after making a complete analysis of the literature, demonstrated its dependence in the vast majority of cases upon sclerosis and obliteration of the smaller arterial branches and terminals, as opposed to Charcot's view that it depended more on the disease of the larger trunks. Since then a few cases have been reported in the United States, but no contribution could be found on the subject in the literature of Great Britain. The inference is that the malady is rare in Great Britain, or has not obtained widespread recognition.

After a detailed description of the etiology, pathology, and symptomatology of the disease, the author describes four cases, illustrating his description with radiographs showing deposits of lime in the posterior tibial and dorsalis pedis arteries. In two cases he finds an underlying neurotic temperament—a point on

which Oppenheim lays stress as a causative factor—and in the other two cases a history of excess in use of alcohol and tobacco, and exposure of the feet to wet and cold over a prolonged period, points of etiology emphasised by Erb.

He concludes that angio-sclerosis of the extremities in its gravest form, uncomplicated by a vaso-motor neurosis, leads to spontaneous gangrene.

If the angio-sclerosis occurs in combination with a vaso-motor instability and a tendency to vaso-motor spasm, the syndrome of intermittent claudication results; characterised by sensory and motor manifestations during functional activity, and a rapid and permanent restoration to the normal during rest. The same condition may be observed in relation to the various organs of the body as in some cases of angina pectoris.

COLIN K. RUSSEL.

**ON THE PLANTAR REFLEX AND BABINSKI'S SYMPTOM IN (262) A THOUSAND YOUNG CHILDREN.** (Ueber den Fusssohlen-reflex und das Babinaki-Phänomen bei tausend Kindern der ersten Lebensjahre.) GOTTFRIED ENGSTLER, *Wien. klin. Wochenschr.*, June 1, 1905, p. 567.

WHEN Babinski published his first observations in 1898, he noted that an extensor response was normally present in new-born children. All subsequent writers on the subject are agreed that this is so, and that Babinski's symptom cannot therefore, in them, be regarded as of any pathological significance. There is still, however, great difference of opinion as to the age at which the extensor normally gives place to the flexor response. With a view to clearing up this point, Engstler has carefully studied the plantar reflex in one thousand children under three years, who were free from cerebro-spinal disease.

He found that these children could be divided into three classes, according as they showed extensor response, flexor response, or no response at all. In a few cases the extensor movement only affected the great toe, or the smaller toes only were flexed, the great toe remaining still; but these irregular cases were classed with the others for statistical purposes. On tabulating his results he arrives at the following conclusions:—

1. In new-born children, especially when they are prematurely born, there is an extensor response almost invariably.
2. In children in the third year of life a flexor response is present, with hardly any exception.
3. During the first and second years the preponderance of dorsal over plantar flexion steadily diminishes. By the end of

the first year the proportion of each is about 50 per cent. of the whole.

4. During the transition period (especially in the second year) the reflex is often absent.

As the result of these conclusions, Engstler agrees with J. L. Morse that Babinski's symptom has no value as a sign of disease in children under two years. His observations, however, do not tend to confirm Kalischer's theory that there is a close connection between the commencement of plantar flexion and the child's learning to walk. He found a flexor response in many children who could not walk at all. In severely rickety children he found the time of transition much delayed; but it usually took place before they began to walk. He is inclined, therefore, to agree with Passini that their backwardness in this matter is due, not to their lateness of walking, but to the general delay of their physical development.

JOHN THOMSON.

**THE REFLEXES OF THE DISTAL SEGMENT OF THE UPPER**  
(263) **LIMB AND THEIR DIAGNOSTIC SIGNIFICANCE.** (*Ueber*  
*Reflexe distaler Abschnitt der oberen Extremität und über ihre*  
*diagnostische Bedeutung.*) W. VON BECHTEREW, *Neurolog.*  
*Centralbl.*, 1905, p. 392.

BECHTEREW enumerates the various reflexes which can be elicited in the upper limb. Of these, the reflexes in the proximal part of the limb are familiar, namely, the biceps and triceps jerks, the scapulo-humeral, the acromion, and the pectoral reflex. In the distal segment of the limb, reflexes may also be elicited by percussion on the lower ends of the radius or ulna. There is also the metacarpo-phalangeal reflex, consisting in a flexion of the fingers when the back of the metacarpus is tapped at its upper end, but this is usually present only in cases of increased reflex excitability. Bechterew also refers to a hand-reflex, elicited on percussion of the ulnaris externus muscle, close above the head of the ulna, and consisting in a movement of extension and abduction of the hand. This also is chiefly found in cases of increased reflex irritability. In such cases also he frequently obtains a reflex in the flexors of the fingers and wrists produced by tapping their tendons just above the anterior annular ligament.

A pronator reflex is also occasionally obtained, consisting in pronation of the forearm when it is supported passively with the elbow semi-flexed and the hand lightly supinated, the tap being delivered on the subcutaneous border of the ulna. The pronation

which results, Bechterew believes to be produced by the pronator quadratus.

The variability of these reflexes, however, renders it necessary to compare the phenomena on the two sides before considering the presence or absence of any one of them to be pathological.

PURVES STEWART.

**THE LIGHT REFLEX IN GENERAL PARALYSIS.** (*Le réflexe (264) lumineux dans la paralysie générale.*) MARANDON de MONTYEL, *Arch. de Neurol.*, June 1905, p. 417.

THIS paper gives the results of the author's examination of the pupil-reflex in 140 general paralytics from the onset of the disease until the death of the patient; the statistics relate to exaggeration, diminution, and abolition of the reflex.

In three-quarters of all cases, the pupils are abnormal. Abnormality occurs very early in the disease, and is present in more than half the cases on first examination. Abolition of the reflex is always preceded by diminution, the abnormality increasing from the first to the second stage, and from the second to the third.

Exaggeration of the reflex occurs in the initial phase only, is always succeeded by diminution, and is never unilateral. In no case are the pupils normal from beginning to end of the disease.

Unilateral abnormality is rarely met with, and, in one-third of such cases, is but temporary. Still more rarely are the two irides abnormal in different ways, and then the difference is but temporary. Unilateral abnormalities of the initial stage sometimes persist in the intermediate stage; but they diminish in frequency and persistence with the progress of the disease, and disappear completely in the third period.

The light-reflex is not always abolished in the terminal phase of general paralysis, it may even be normal; but any alteration of the reflex is then of the same nature in the two eyes.

W. H. B. STODDART.

**APFOLEMENT BULBAIRE.** LÉOPOLD LÉVI, *Presse Méd.*, July 12, (265) 1905, p. 433.

BULBAR reactions are functionally normal when a given centripetal stimulus produces an adequate centrifugal response—*e.g.* when a normal, physiological hunger is the consequence of the appeal addressed by the totality of the gastric mucosa to the centre for diastatic activity. But apart from permanent lesions, it is not

infrequent to find bulbar centres so modified by a variety of circumstances that a normal stimulus produces a supernormal result: an insignificant emotion betrays itself by tears, tachycardia, even diarrhoea. This disproportion between excitation and reaction is one of the features of "bulbar neurosis."

Many bulbar syndromes are of course systematised, migraine, *e.g.*, being considered one of these symptom-complexes, but when one remembers the remarkable concentration and condensation of nerve centres in the bulb, it is easy to appreciate the variety of reactions pathologically possible, apart from systematised disease of tracts or fibres. Such conditions are simply those of "bulbar disarray"; for an instance, the association clinically of hunger with syncope, anxiety, migraine, amblyopia, etc.

A special type of "bulbar disarray" the author describes under the term "affolement bulbaire." [When a magnetised needle in the proximity of iron, or under the influence of a storm, an aurora borealis, etc., undergoes sudden irregular deviations, it is said to be "affolée," and the word is also used to describe the "infatuation" which makes a lover capable of mad acts.] By this expression he signifies sudden irregular transient bulbar disorders, accompanied with a certain degree of terror, or rather anguish.

His patient, 35 years old, on the twenty-sixth day of a severe typhoid fever, suddenly had thrombosis of the left internal saphenous vein, and this was ushered in with tachycardia and a thready pulse, anxiety, fear of impending death, and vasomotor phenomena, profuse perspiration, paroxysmal dyspnoea, paroxysmal thirst, and vomiting, the symptom-complex lasting about twelve hours. Fourteen days later a fresh thrombosis was accompanied by the same phenomena, with shivering and rigors in addition.

It is probable that the thrombosis produced instant modification of circulatory equilibrium, a sort of shock to which the bulbar cardiac centres at once responded. It is interesting to note that what is known clinically as paroxysmal tachycardia is sometimes associated with præcordial distress, perspiration, vomiting, and reduced blood-pressure. The condition of anguish, with a sensation of impending death, was a marked feature in the author's case. These phenomena are frequently associated with dryness of the throat, paroxysmal thirst, and further respiratory, circulatory, and vasomotor disorders. They are the result of irritation of the vagus in some part of its distribution, which by central spread leads to disturbance of functions essential for existence; they are accentuated by the reaction of the modified circulation upon the centres themselves. Occurring as they do in innumerable conditions of shock, one may regard these symptoms as indicating a process of defence on the part of the organism.

S. A. K. WILSON.

**INTENTION TREMOR LIMITED TO THE LEFT UPPER EXTREMITY.** (Tremblement intentionnel limité du membre supérieur gauche.) BOUCHARD (Lille), *Journ. de Neurol.*, April 20, 1905, p. 147.

THE author describes the case of a man æt. 62, in whom, after a sudden attack of giddiness, in which he fell down and almost lost consciousness, there developed a tremor in the left arm, on any attempt to use it, characterised by wide oscillations affecting the whole limb; these were not increased on closure of the eyes. They were not irregular like chorea or athetosis, but more pronounced at the proximal than the distal segments, and augmented with the extent of the movement and the closer the hand came to the object desired. He could not hold his extended hand steady, nor keep his index finger on his nose. If the forearm was held, the hand showed the movement, but more feebly, and if the hand was held there were occasional movements in one or two fingers. It ceased only when the limb was perfectly at rest. There was no weakness in the grasp, but some stiffness in using the hand.

Three months later he had another similar attack, followed by difficulty in speaking, and his right arm was affected with an intention tremor similar to the left, but this only lasted a few hours. That in left arm remained present till death, two years later.

At autopsy there was found a slight atheromatous degeneration of cerebral vessels, and an area of softening was discovered in the internal capsule on cutting the brain in that part of the right hemisphere exposed by Pitres' pediculo-parietal cut passing immediately behind the lenticular nucleus. It extended "from above downwards and from in outwards from the upper part of the optic thalamus to the retro-lenticular region. It was characterised as irregular, with the appearance of a mild softening, well defined above, where its extent was about two millimetres, while below it became diffuse." It would be interesting to know if the brachium conjunctivum was involved in the diffuse part of the lesion. As it would be in such close proximity, it seems possible. According to G. M. Holmes (*Brain*, Autumn 1904), a lesion involving the superior cerebellar peduncle before it entered into the red nucleus on the right side would account for just such a tremor in the left arm.

There is nothing noted which would account for the second attack with transient tremor of the right arm.

In support of his view that the tremor was caused by an irritating lesion of the pyramidal fibres to the upper extremities, the author quotes two cases. The first, reported by Bennett and Campbell in 1885, a hæmorrhage into the internal capsule, causing



paralysis of right side of face and right arm, with temporary loss of speech; but the tremor present in the arm is not described. The second, reported by Demange in 1883, in which the lesion is so large that nothing definite should be concluded from it.

COLIN K. RUSSEL.

**LIMITED RETRO-ANTEROGRADE AMNESIA COMMENCING (267) SUDDENLY AFTER A STROKE IN A CASE OF GENERAL PARALYSIS.** (*Amnesie localisée rétro-antéro-grade ayant débuté brusquement par un ictus chez un paralytique général.*) ROY and DUPOUY, *Rev. Neurolog.* June 15, 1905, p. 578.

As a general rule troubles of memory in general paralysis of the insane are essentially diffuse, progressive, unsystematised. They are one of the most precocious and striking features in the generalised psychic enfeeblement so characteristic of the disease. The failure of memory of recent events contrasts with the conservation of remote incidents, but the progress of this retrograde amnesia soon obliterates all souvenirs, early or late: fixation of recent occurrences becomes quite impossible; their localisation, retention, and reproduction soon are quite absent. The authors are able to report a case offering conspicuously differing features from the usual, in that the troubles of memory came on suddenly as by a stroke, and remained localised strictly to the last twelve years of the patient's life: a vanman in Paris, he believed himself to be still a butcher at Brunoy; divorced and married a second time, he persisted in considering his second wife his first, and her children his first wife's children; an orphan for twelve years, he never ceased lamenting the trouble he was giving to his "poor old parents," and wrote them letters full of filial affection, etc. It is evidently a pure instance of localised and limited amnesia, bearing a close resemblance to traumatic amnesia, a phenomenon not by any means common in a disease essentially progressive.

S. A. K. WILSON.

**ABOUT DREAMING, LAUGHING, AND BLUSHING.** By Sir (268) ARTHUR MITCHELL, K.C.B. William Green & Sons, 1905.

THE author deals with the subject of dreaming, laughing, and blushing in a philosophic and interesting manner. He holds with Sir Benjamin Brodie, John Reid, and others, that thinking continues during sleep, in dreams, and, indeed, only ceases with life. It is argued that the difference between thinking while

awake and during sleep is that in the former case the thoughts are directed by the will into definite channels, while in the latter case they occur at random. The less the thoughts are under the guidance of the will, the more does the condition approach to the state of dreaming, for which reason those who have little control over their thoughts are apt to fall into "day dreaming." We are inclined to doubt whether the author is correct in assuming that directly an individual ceases to exercise control over his thoughts he is practically asleep. It seems to us that the awareness of an external world round and about us constitutes the criterion of awakeness. Not until he is unconscious of the external world can anyone be considered asleep, and it is possible for an individual to allow his thoughts to be passively directed by external occurrences, for him to be, *i.e.* in a wholly objective state of mind—wholly uninfluenced by that illusive quantity termed the will—and yet remain completely awake.

The author holds that the sustained effort of the will in thought causes fatigue, and that dreaming, needing no effort of will, is a mental recreation inducing no fatigue. He mentions a fact of some interest to the neurologist, *i.e.* that dreams and hallucinations may occur during momentary spells of sleep, and contends that many of the spectres we hear of are only dream hallucinations. A person falls asleep for a moment, dreams of someone, and on suddenly waking up believes—all unconscious that he has been asleep—that he has actually seen an apparition.

It is pointed out that dream thinking is rapid and incoherent, passing from one subject to another, and from one place to another, without causing surprise, the most incongruous occurrences exciting no wonder; and it is contended that dreaming and delirium are closely allied. Another feature of dreaming is that during it the moral sense is in abeyance: "a kind-hearted person may commit a cruel, an honourable person a base, and a virtuous person an immoral deed in sleep, without feeling that anything wrong has been done."

Laughter, the author regards as a transitory state of mental disorder, for no one can think or speak coherently, or rationally during a convulsive fit of uncontrollable laughter. He points out that it may be the expression of several different states of mind, not only of joy, but of mean, contemptible, and even cruel emotions; and that laughter so arising is only pardonable on the view that it is a state of mental disorder.

The writer insists that as in dreaming and laughing, so in blushing, the mind is for the time being disordered, as is expressed by the phrase "covered with confusion," as applied to the blusher.

HARRY CAMPBELL.

## PSYCHIATRY.

**STATISTICS IN INSANITY: A UNIVERSAL SCHEME.** C. C.  
(269) EASTERBROOK, *Journ. Ment. Science*, April 1905, p. 348.

THE Lunacy Blue Books and the Annual Reports of asylums and similar establishments for the insane furnish the Annual Medical Statistics, and so are our main source of information from year to year, as to the production of insanity in individuals and in the community and country generally, and as to the results of the institutional care and treatment of the insane. It is important, therefore, that the system of annual statistics employed should be accurate and illuminating in the eyes of the statistician, lawyer, and physician; and further, that for comparative purposes the system should be equally applicable by the medical authorities of any individual asylum or similar institution for the insane in this or any other country, and by the central lunacy authorities of different countries, notwithstanding variations in lunacy legislation and administration. This paper, which shows the possibility of such a system, is divided into three parts.

I. The Methodology of the Annual Statistics of Insanity suitable for Asylums and similar Institutions.

The following terms and their definitions are necessary for clearness:—

An *Insane Person* is a person certified as insane. Insane persons are to be clearly distinguished from (1) Borderland or unconfirmed cases of insanity, who live, as in Scotland, in private houses in the community with the sanction of, and subject to the supervision of, the central authorities, but who, being uncertified, are not "insane persons." (2) Voluntary Boarders, who live in asylums and similar institutions, but who, being similarly uncertified, are technically not insane, that is, in the eyes of the law they are sane persons. All certified insane patients, it is particularly to be noted, continue to be "insane persons" so long as they remain unrecovered, and it is only when they are pronounced recovered that they are released from their certificates of insanity and so cease to be insane persons, and consequently come to be regarded by physician and lawyer as sane and free agents in the community as they formerly were before certification.

*Institutions or establishments for the insane* are for the institutional care and treatment of the insane, with a recognised medical and nursing staff in attendance, and include—asylums, registered hospitals, licensed houses, idiot and imbecile institutions, lunatic workhouses or lunatic poorhouses or lunatic departments of the same, lunatic State prisons or lunatic departments of the same,

lunatic departments of State military and naval hospitals, and colonies for insane epileptics and other lunatics.

*Dwellings for the insane* include unlicensed houses for single certified patients, and in Scotland "specially licensed houses" for two, three, or four insane patients; and in England or Ireland any similar provision which may arise in view of recent or future legislation which will enable a similar small number of lunatics to be gathered together under one roof for the enjoyment of home care, as distinguished from institutional care and treatment.

A *home care* is an insane person who either passes to (admission) an establishment from the community (in which he has been living in a "dwelling," or perhaps with friends or relatives, or even in his own home, and whether he has been kept "for profit" or not); or who passes from (discharge) an establishment to the community (to live in a "dwelling," or with friends or relatives, or even in his own home, and whether to be kept "for profit" or not).

A *transfer* is an insane person who passes from one establishment to another in the same country. He is, of course, "discharged" from the one establishment and "admitted" into the other.

A *transference* is an insane person who passes from (discharge) an establishment in one country to (admission) an establishment in another country. It is to be noted that the home cares—transfers and transferences—are all insane persons; that is, at some time previously they have been certified as insane, and have not as yet recovered, and so have not been released from their original certificates of insanity; but nevertheless the home cares living in the community cannot be admitted into an establishment without being certified again, and similarly transfers and transferences cannot pass from establishment to establishment without proper sanction from medical certificate and judicial or lunacy authority.

A *re-admission* of the current year is a person admitted more than once during the said year, and is dealt with statistically as at the time of his first admission during that year.

A *pre-discharge* of the current year is a person discharged more than once during the said year, and is dealt with statistically as at the time of his last discharge during that year.

*Persons discharged* fall into two distinct groups—

(1) Recovered, or direct discharges; that is, persons who return to the community as sane and free agents, released from their certificates of insanity.

(2) Unrecovered, or indirect discharges; that is, persons who leave an establishment still as insane persons—they may be improved ("relieved") or unimproved ("not improved")—and

who are home cares or transfers or transferences, according to their destination.

*Persons admitted* fall into two distinct groups—

(1) Indirect admissions; that is, already insane unrecovered persons coming from the community as home cares (these are admitted usually on account of some exacerbation of their insanity), or coming from establishments as transfers or transferences (these are admitted usually for administrative reasons, or for the sake of a change to some other institution). In all cases, medical recertification and renewal of the proper judicial or central sanction are necessary for the admission of such "insane persons."

(2) Direct admissions, that is, persons admitted on account of the onset of a distinct attack of insanity, in consequence of which certification has become necessary for the first time in relation to this attack, and perhaps for the first time in the patient's life. For statistical purposes, therefore, direct admissions may be differentiated into: (a) Persons with no previous attack; (b) Persons with one or more previous attacks; and (c) Persons in whom it is unknown whether the attack is the first or not. It is obvious that the direct admissions, especially the first attack cases, form *the* important group of admissions for purposes of statistics bearing on the growth of insanity in a community, and that to include amongst them any of the indirect admissions as above defined is to introduce insidious fallacy and misrepresentation of truth amongst the facts upon which are based our investigations into the causation of insanity, the production of insanity in communities, and such like lines of inquiry.

With these explanations, it will be seen that for purposes of annual statistics of insanity in asylums and similar institutions, accuracy is secured: (1) By dealing only with certified insane patients, voluntary boarders being excluded from the medical statistics. (2) By reducing cases admitted and cases discharged to terms of persons admitted and persons discharged, namely, by eliminating the re-admissions and pre-discharges of the current year; there is obviously no necessity to reduce cases to terms of persons in the case of those who die during the year, or in the case of those on the registers on the last day of the year. (3) By differentiating the persons admitted into direct admissions and into indirect admissions (home cares, transfers, and transferences), and the persons discharged into recovered, and unrecovered (home cares, transfers, and transferences). The statistical groups of persons to be dealt with, therefore, comprise the following:—

(1) Persons admitted: (a) direct; (b) indirect.

(2) Persons discharged: (a) direct or recovered; (b) indirect or unrecovered.

(3) Persons dead.

(4) Persons remaining on the registers on the last day (31st December) of the year reported upon, who are not included in the above group (1).

II. The Medico-Statistical Data of Insanity, and their embodiment in a Medical Register of insane persons. A register is submitted which contains in its thirty columns provision for all the medical data of insanity likely to be required for the complete study in private of one's patients; and for the purposes of the annual statistics of asylums and similar institutions, certain of these data are selected in relation to each of the above statistical groups of persons, only those data being chosen which are likely to give useful and reliable information. Thus in the case of the *direct admissions*—the most important group in regard to our information about the production of insanity in individuals and in the community—the data suggested are:—Sex, age, conjugal state; ? nationality and ? religion; education and occupation; hereditary potentialities; occurrence of previous attacks, with age at first attack; duration of illness on admission; etiological factors; suicidal and homicidal tendencies before admission; mental state on examination; bodily state on examination; diagnosis and prognosis. In the case of the *indirect admissions*, the only data of interest—and probably not of much importance medico-statistically—are age, duration of illness, mental and bodily states, diagnosis and prognosis. It is recommended that separate medical admission registers—one for each sex—be kept, and that the direct admissions be entered in black ink and the indirect admissions in red ink. In the case of the *recoveries*, the important data to know are the age on recovery, the duration of the illness (including duration before admission, length of residence, and total duration), and the diagnosis of the insanity from which there has been recovery. In the case of the *unrecovered*, hardly any medical statistics of value can be obtained, so many patients being discharged for administrative and non-medical reasons.

In the case of the deaths, the chief medical data for statistical purposes are: the age at death, the cause of death (and whether ascertained by post-mortem examination or not), the duration of the insanity (differentiating duration before admission, length of residence, and total duration), and the diagnosis (nosological) of the insanity. It is recommended that the medical data in connection with the discharges and deaths be incorporated in the existing civil registers, no special medical registers being necessary for the comparatively few data required. In the case of the *persons remaining* at the end of the year, the data suggested are the age, total duration of the insanity up to date, diagnosis and prognosis as to recovery or improvement.

III. The Construction of Medico-Statistical Tables for the annual statistics of insanity in asylums and similar institutions. The headings and titles of twenty tables are submitted, showing how the above-mentioned data requisite in connection with the various statistical groups of persons may be most conveniently arranged in tabular form, and correlated where advisable so as to bring out the greatest degree of medical instruction and utility.

AUTHOR'S ABSTRACT.

**A CLASSIFICATION OF THE LITERATURE OF INSANITY  
(270) WITH RELATIVE INDEX, ON DEWEY'S DECIMAL  
SYSTEM.** A. R. URQUHART, *Journ. Ment. Sc.*, April 1905.

IN this paper the author illustrates in a practical manner how Dewey's decimal system of classifying the books in a library may be applied in the case of the literature pertaining to mental diseases.

It is a great gain in using Dewey's classification that one is at once on familiar terms with all the books and shelves thus arranged, wherever they may be.

H. DE M. ALEXANDER.

**ON THE MENTAL DISORDERS DUE TO ARTERIO-SCLEROSIS  
(271) AND THEIR RELATIONS TO THE SENILE PSYCHOSES.**

(Ueber die Geistesstörungen bei Arteriosklerose und ihre Beziehungen zu den psychischen Erkrankungen des Seniums.)

Prof. BUCHHOLZ (of Hamburg), with 3 plates, and 6 figures,  
*Arch. f. Psych. u. Nervenk.*, Bd. 39, H. 2, 3.

THE author gives the detailed observations and pathological findings in five cases of insanity on the basis of marked arterio-sclerosis. The clinical picture varied greatly; in one case the diagnosis of general paralysis had been made, in other cases the symptoms were more suggestive of senile dementia.

The lesions disclosed by microscopic examination were neither uniform in localisation nor in their nature; in one case the brain would be the seat of the most advanced change, in another case the cord or the brain-stem; similarly the grey matter would be most damaged in one, the white matter in another case. In addition to the ordinary foci of softening and hæmorrhages, various other histopathological processes were demonstrated—purely sclerotic processes, perivascular gliosis with disappearance of ganglion cells, chronic subcortical encephalitis (Binswanger), various types of cavity formation, and in addition changes due to

the pressure of arterio-sclerotic vessels upon the surrounding nervous tissue. The variety of the pathological processes, taken into consideration along with the general disturbance of nutrition, explains the variety of the symptoms found in arterio-sclerotic insanity. While Binswanger and Alzheimer have insisted upon certain characteristic types of arterio-sclerotic brain degeneration, the majority of the cases are too complicated to be brought under these types: in the present state of the subject detailed observations and results of pathological examination are to be desired.

C. MACFIE CAMPBELL.

**ON THE SYMPTOMATOLOGY OF MELANCHOLIA.** (Zur Symptomatologie der Melancholie.) O. JULIUSBURGER, *Monatsschr. f. Psych. u. Neur.*, May 1905, p. 389.

THE author discusses certain symptoms in a woman suffering from a second attack of melancholia. The patient felt to a certain extent as if her personality were divided. "I have two I's . . . the one sits in the head, the other here (in the breast) . . . the one has no thoughts, only hears and takes up what it hears from others, the other is anxious to be up and doing." Juliusburger tries to explain the condition by the assumption of the "sejunction" or dissociation of two spheres, the one including all somatic sensations, the other including the mental processes involved in one's consciousness of the world and of one's own personality. He quotes the patient's words: "I do not *hear* what is said, but I understand the sense of it": such a statement may be explained by the assumption of a certain want of the organic sensation peculiar to the sense organ, while the further elaboration of the sense impression is not affected. The dissociation from the elaborating spheres of organic sensation, which is so closely connected with the emotional and volitional life, would, if the intellectual elaboration of impressions were intact, lead to a want of harmony between the sentient and intellectual functions similar to that expressed by the patient when she said, "I have two I's."

C. MACFIE CAMPBELL.

**CONTRIBUTIONS TO THE SUBJECT OF PARANOIA.** (Beiträge zur Lehre von der Paranoia.) M. FRIEDMANN (of Mannheim). *Monatsschr. f. Psych. u. Neur.*, May, June 1905, p. 467.

THIS first contribution deals with mild forms of paranoia. Friedmann first discusses a small group of cases which run a favourable course, and which seldom need to enter an asylum. He gives the



records of five patients belonging to this group, the age of the majority being between 30 and 40, and four out of the five patients being women. In all cases there was a neuropathic or psychopathic heredity. They presented a striking similarity in their clinical course. In each case under the influence of some mental shock or worry—the failure of a last chance of marriage; the thought of having committed a slight indiscretion; the accusation of disloyal behaviour to comrades, the suspicion aroused by a letter wrongly delivered; the knowledge that both ovaries had been removed—the patient developed delusions which remained restricted to the exciting cause and to the persons connected with it.

There was present in all cases the idea that patient was being watched. After a period varying from one to two and a half years the condition was practically cured, but there was no insight; the emotion connected with the event had faded, and the cure was the same as having outlived an actual occurrence. No hallucinations were observed; the condition was improved by the withdrawal of the exciting cause, and sometimes by advice. The effect which characterised the cases was one of despondency and extreme irritability.

This clinical picture is called by Friedmann, "mild systematizing paranoia"; psychologically it is characterised by the endogenous origin of the delusions which develop in a logical manner from real worries, the false element being the judgment of value of certain facts. This distorted judgment has its origin in the disposition of the individual, her over-sensitiveness, obstinacy, and egotism; usually there is a bad heredity. The intimate connection of the delusions with the core of the patient's personality is seen in the nature of the cure; the distorted product of thought remains a permanent mental acquisition. For these reasons the author applies the term endogenous to such delusions.

He next reports shortly six cases of paranoid attacks, some of them being paranoid forms of manic-depressive insanity, where the delusions were exogenous; they did not develop secondarily, by a process of logical reasoning from some definite cause of worry, but sprang up immediately on the basis of a morbid affect. In these cases there was no true continuity of the delusions with the normal thought of the individual. The beginning of the attack was usually sudden, and recovery where it occurred meant a return to the previous thought of the individual.

Friedmann agrees with Wernicke that at present we can only establish a number of clinical types of paranoia; the present rubrics are quite inadequate for the complexity of clinical facts. All the more is it necessary to give definition to our clinical types, and Kraepelin has taken a step in this direction in excluding from his type of paranoia the paranoid forms of dementia præcox. He

uses this latter, however, as a pot into which a large number of complex forms are thrown.

The author makes some remarks upon the nature of the character which predisposes to a paranoid development; he promises a more detailed description later.

A specific mental predisposition to a paranoid development *per se* is rare; where a person develops a simple delusional system, not shared in by his environment, there are usually other marked traits of mental inferiority associated with the delusions.

Still such cases do occur. Friedmann records four cases showing development of delusions on the basis of a peculiar affective character, without further implication of the intelligence. These four cases differed from true chronic paranoia by their episodic nature and through the absence of ideas of reference; and in this latter respect from "mild systematising paranoia."

One of the cases is especially interesting, being a very short attack of ideas of jealousy with no alcoholic nor epileptic etiology, but springing simply from a condition of general nervousness.

It is impossible to draw a hard and fast line between similar cases and affective anomalies of character which we cannot call pathological.

C. MACFIE CAMPBELL.

**CLINICAL CONTRIBUTION TO THE SUBJECT OF KRAEPE-**  
(274) **LIN'S DEMENTIA PRÆCOX.** (Klinischer Beitrag zur Lehre von der Dementia præcox Kraepelins.) A. SCHOTT (of Zwiefalten), *Monatsschr. f. Psych. u. Neur.*, Bd. 17, Ergänzungsheft, 1905.

TWENTY-FOUR cases of dementia præcox, grouped according to the diagnosis made before admission to the asylum. In slightly over half of the cases there was bad heredity, which was most marked in the catatonic and paranoid forms. The author in conclusion discusses the general clinical picture of dementia præcox.

C. MACFIE CAMPBELL.

**ON AUTO-INTOXICATION PSYCHOSES.** (Ueber Autointoxicationen.) E. MEYER (of Königsberg), with 1 plate, and 3 charts, *Arch. f. Psych. u. Nerven.*, Bd. 39, H. 1.

MEYER reports 8 cases of psychoses which developed apparently on the basis of auto-intoxication: the mental symptoms were insufficient to make the diagnosis without the presence of bodily symptoms, among which headache, dizziness, fainting, and tremor were the most important nervous manifestations, while the general condition of the patient showed a severe affection without very

definite cause. Careful studies of the metabolism of the patients were not made, but Meyer calls attention to the frequent difficulty in making these, and to the uncertain value of indican estimations in proving or disproving auto-intoxication. The mental condition of the patients was a dreamy confusion with incoherence, difficulty in comprehension, tendency to perseveration and stereotypy, and often varying hysteriform features. Meyer concludes that the mental condition is so similar to that seen in other toxic and exhausted conditions, that we cannot talk of a specific auto-intoxication psychosis: it is the physical condition which enables us to diagnose the cause, and justifies the name.

C. MACFIE CAMPBELL.

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## Obituary

### CARL WERNICKE.

A SHORT time ago, as the result of a deplorable accident, there took place the sudden death of Professor Carl Wernicke, Director of the Psychiatric Clinic in Halle.

Carl Wernicke was born on May 15, 1848, at Tarnowitz, in Silesia. Breslau, however, was the real scene of his activity. There he finished his studies, and became assistant at the City Asylum. He left Breslau for a time only, going to Berlin in 1875, where, till 1878, he acted as first assistant at the Psychiatric Clinic at the "Charité," under Westphal. In 1885 he returned to Breslau, filling up to 1904 the post of Professor and Director of the Psychiatric Clinic in that town. In 1904 he went to Halle. He felt his departure from Breslau very keenly, having no foreboding that his parting from life was so soon to follow.

The fatality which swiftly carried Wernicke away was an extremely tragic one. Whilst on a holiday tour he was run over by a waggon, and his right lung was injured. Thus died Wernicke, faithful to his scientific vocation up to the last moment. Even in the two closing days of his life, whilst in great pain and feeling the approach of death, he lay working at the second edition of his "Grundriss der Psychiatrie," sometimes writing with his own hand, sometimes dictating till his voice failed him. Repeatedly he allowed himself to be called back to consciousness by the artificial inhaling of oxygen, until at last death snatched him away for ever. His death was heroic as his life was scientific.

It seems almost superfluous to enter more fully into the question of Wernicke's value to science. This value lies not only in the great number of original discoveries, investigations, and writings which we owe to him. I mention only his epoch-

making work, "Ueber den aphasischen Symptomencomplex," his text-book on "Gehirnkrankheiten," in three volumes (1881-1883), his works on polio-encephalitis hæmorrhagica superior and hemianopic pupil-reaction, his exhaustive investigation into the type of paralysis in hemiplegia, his "Grundriss der Psychiatrie," and his "Atlas des Gehirns in photographischen Originalien," which he published along with his assistant; but still more than in these works, monumental though they be, Wernicke's value to our science lies, in my opinion, in the absolutely original systems which he did so much to create. He was one of the first to define sharply and clearly the conception of localising symptoms in brain disease, to recognise distinctly the contrast between these localising symptoms and general signs, to lay stress upon these, and to explain them in detail. Wernicke was, in point of fact, one of the creators of our present system of cerebral pathology, helping in this way to raise the study of localisation to its true significance.

Equally original and creative is his system of psychiatry. Mental diseases are cerebral diseases; they submit to the laws which govern the nervous system generally and the brain in particular. In these, also, the general symptoms are to be clearly distinguished from the localising symptoms. This point of view Wernicke carried out consistently into details. His method of considering the symptoms of mental disease was formed by the application of the physiological and pathological data of the brain, and in this connection, so long as there exists a science of neurology and psychiatry, the name of Wernicke will never die.

The Editors' thanks for this Obituary Notice are due to Dr Otfrid Foerster of Breslau.

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# Review of Neurology and Psychiatry

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## Original Articles

### THE GROWTH OF HISTOLOGIC TECHNIQUE DURING THE NINETEENTH CENTURY.

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(Continued from p. 515.)

#### (B) Cellular Elements.

WITH the appearance of WEIGERT's method, fibre anatomists were provided with a staining procedure whose results, as compared with previous technique, seemed to leave little to be desired. Cell-histologists were, however, still at a disadvantage. Carmine, nigrosin, hæmatoxylin were still widely used; the GOLGI silhouette method had indeed revealed external contours and furnished remarkable pictures of dendritic and axis-cylinder courses, but as yet there was no specific *histologic* cell stain.

This to the fibre anatomists of the seventies seemed a matter relatively unimportant, judging from the viewpoint of MEYNERT, that the study of the internal morphology of ganglion cells would contribute but little to advancing the knowledge of nerve centres, and that external relations and connections made up the all-important subject. Small wonder that the method of GOLGI, which satisfied exactly these conditions, enjoyed such a remarkable prestige, or that its value was over-estimated.<sup>1</sup> Only

<sup>1</sup> Over against the indisputably great service which the silver impregnation method has rendered to the study of the central organs, stand out no less conspicuously its limitations and dangers. Its inconstancy is notorious. It often

gradually did the reaction develop, bringing back the attention of brain anatomists to the investigation of ultimate cytologic details, such as had busied the workers of an earlier period, and thus emphasising the necessity of improved methods of cyto-histologic demonstration.

The method which made possible the study of the finer internal structure of the cellular elements of the central organs—a study which dealt at first only with the nervous elements, but which now includes both ganglion and glia cells alike—was the one discovered by NISSL, and which bears his name. The NISSL method, like the majority of epoch-making discoveries, was the result of years of experiment, during which it passed through various stages, and provisional results were from time to time made public. Some of these, though no longer orthodox, are still current in the text-books. Moreover, modifications and “improvements” almost without end have been proposed or unconsciously substituted by various observers, until the term “NISSL method” has come to have an extreme elasticity of meaning. There is indeed a degree of justice in the remark of LUGARO that strictly the NISSL method is practised only by NISSL himself and his pupils. When the number of the latter is taken into account, however, it will be seen that the use of the authorised method is sufficiently widespread. Forming as it does one of the most important chapters in the history of histologic procedures, a brief review of the principal steps in its development will not be without interest.

Early in the eighties, while assistant at the Jusam Hospital in Frankfort a. M., NISSL first turned his attention to improving the means hitherto available for studying the nerve cell. Like other brain histologists, he had been accustomed to work with Müllerised material stained with carmine. For the purposes in question this method obviously left much to be desired. First, he tried the effect of substituting the oil immersion lens for the usual high powers commonly in use. This was the first step in the right direction, although there was but little gain in detail from carmine preparations. It was evident that the defect lay in the staining method itself.

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renders impossible the differentiation between ganglion cells and glia elements. By its use GOLGI himself discovered only slight cyto-architectonic differences between occipital and motor cortex (1).

About this time FLEMMING's work on the structure of the spinal ganglion cells appeared (1882), and NISSEL, well pleased with its beautiful illustrations, became more deeply impressed with the necessity of an appropriate method by which these studies could be extended to the cellular elements of the central organs. He began a series of comparative experiments, using various stains, Müller's fluid being still the hardening medium. Magenta red was at length adopted as the stain of choice, and it was further discovered that more satisfactory results could be obtained with watery solutions than with alcoholic. The magenta pictures were often particularly fine, and offered a striking contrast to the previous carmine preparations.

Continuing his experiments, NISSEL next made trial of different hardening media, and soon came to a realisation of the important fact that *for fixing and hardening nervous tissue preparatory to staining its cellular constituents, alcohol is to be preferred to all other media*. The method was first published in 1885, and consisted of the following steps:—

- (1) Hardening in 96% alcohol.
- (2) Staining with magenta red.
- (3) Decolourising in alcohol.
- (4) Clearing in oil of cloves.
- (5) Mounting in balsam.

It was found that material hardened in alcohol could be directly mounted upon a block of wood or cork by means of an adhesive substance, such as gum acacia, and cut at once, thus obviating the delay of an imbedding process, and more important still, the resulting artefacts. This is a point of very great moment, which has not, unfortunately, in all quarters received the consideration it requires. There will be occasion to refer to it again.

For a time the method of 1885 gave excellent results, and apparently satisfied every need, when finally the stock of magenta became exhausted. A new supply was obtained, but of a different and cheaper quality, which proved to be entirely unreliable. There was no longer any certainty as to how the stain would work, or indeed that it would work at all. On Sunday, perhaps, one would obtain superior preparations, and on Monday have no success whatever, all the stain coming out in

the alcohol or oil; again, the glia cells would take up the colour while the ganglion cells were entirely bleached. A third supply of magenta was at length tried, this time of a quality similar to the first, and again the results were more satisfactory. Of all this experience, however, the idea has been born that a method must be worked out which with uniform technique would give absolutely constant results, one which would yield comparable microscopic pictures in *every* case—pictures, indeed, which in normal tissue could be described in advance.

For the experiments NISSL took healthy rabbits, which were killed, and their cortices fixed at once in 96% alcohol. Freshness of material was of course the first requisite, and to this end it is not improbable that the sequence—killing, removal of cortex—may sometimes have been reversed. For staining the sections various of the sulphur-group of synthetic dyes were next tried—toluidin blue, thionin, methylene blue, Dahlia, and others, and it was found that for clear sharp pictures, with completeness of delicate detail, methylene blue excelled all other stains. The question which next presented itself was the strength of the solution to be used. Beginning with a saturated aqueous solution, various dilutions were also tried and their results compared, when it soon became apparent that weaker solutions revealed histologic minutiae more accurately than stronger ones. A 0.375% solution was finally fixed upon as the most favourable, the staining process being promoted by means of heat. The technique was still similar to that in the original publication, merely substituting the dilute methylene blue solution for magenta red.

Preparations made in this manner were brilliant, but had the unfortunate defect of soon fading. Moreover, the oil of cloves, with which the sections were treated, after the excess of stain had been removed in alcohol, continued to draw out the colour. In the case of the intensely stained magenta specimens this had not been a disadvantage, but became so when the more delicate diluted methylene blue solution was used. Origanum oil was somewhat more satisfactory than oil of cloves, but also tended to bleach the preparations, especially if not absolutely pure. Celerity was obviously the keynote of the process. The idea therefore suggested itself of adding the oil to the alcohol, thus accomplishing largely in a single step the processes of decolourising, clearing, and dehydrating. Aniline oil (puriss alb.) was

tried and found good, and a standard decolourising solution was finally prepared by adding 10 c.c. of aniline oil to 90 c.c. of 96% alcohol. Through this sections could be hurriedly passed without losing too much colour. In addition, the subsequent treatment with oils became less essential and could be materially shortened.

The method as thus perfected was more satisfactory than at any previous stage. Still, however, the preparations were not permanent. The next improvement, by which this defect was considerably lessened, was the result of an accident. In an interview with the bacteriologist FRANCK, of Wiesbaden, the latter had related to NISSL how for a long time he had tried without success to give a permanent stain to anthrax spores. When one day several slides, which had accidentally been thrown into the sink, where they had come into contact with soapy water, were examined, and the stain was found to be decidedly improved. Acting upon this hint, NISSL added soap to his staining solution and observed that the results were surprisingly better. The fatty acid from the soap had evidently given the stain a colloidal character which rendered it more intense and permanent. Venetian soap was chosen on account of its purity, being composed of pure olive oil.

Finally, in 1890, after nearly ten years of experiment, came the publication of the perfected method as at present known. Sections were stained with heat in the following solution <sup>1</sup>:—

Methylene blue (B. patent)	.	.	3.75
Pure Venetian soap	.	.	1.75
Aq. dest.	.	.	1000

discoloured rapidly in aniline-alcohol, blotted on the slide and cleared briefly with oil of cajeput, removing the alcohol, covered with benzine<sup>2</sup> to remove the oil, and mounted in balsam, heat being used to expel traces of benzine. Canada balsam, in which the stain tended to diffuse, was finally replaced with colophonium, prepared in the laboratory by dissolving the resin in benzine, and

<sup>1</sup> The stain was found to improve with age. Two bottles are therefore kept constantly on hand, one of which is ripening while the other is being used. The bottles are never washed on refilling, and are shaken well before using.

<sup>2</sup> C.s. xylol may advantageously be substituted for benzine. Its effect is the same, but it evaporates less rapidly, so that there is no danger of the section drying. The need of extreme haste in the final step is thus obviated, and the washing with xylol can be thoroughly and leisurely accomplished.

which, being neutral, did not injure the specimen. Later, xylol-colophonium, which is somewhat more readily prepared, was substituted. Colophonium has the advantage over balsam not only in better preserving the colour of the preparation, but also because it hardens almost at once, and slides can therefore be more conveniently handled. Further, the colophonium does not subsequently run when exposed to heat, and the slides are thus available for projection. Balsam preparations, on the other hand, hardly endure the arc light.

The history of the NISSL technique has been given somewhat in detail, as it is this method which has been the moving force in the unfolding of the modern cyto-histologic period. It is the method which, either in its authorised form or in some of its modifications, employing methylene blue and related dyes, is relied upon before all others in the study of histologic and histopathologic details of the cellular constituents of nervous tissue.

In this connection there are several points of technique which require further mention. WEIGERT, in his fifth and sixth postulates, stated that a satisfactory staining method should consume as little time as possible, and should not injure the specimen. Artefacts, other than those directly conditioned, by the essential steps in hardening and staining, which are thus easily reckoned with, are of course to be kept out as far as possible. Celerity and simplicity of technique, with a minimum of manipulation, should therefore be the histologist's guiding principle. This being recognised, the place of alcohol as a hardening agent is at once apparent. The alcohol method is the only one by which fresh material is carried by a single step from the organ to the microtome (excepting, *e.g.*, the processes of freezing, and of cutting unimbedded Müllerised tissue,—manœuvres which have special and limited application). It is not only absolutely *tuto*, but also pre-eminently *cito et jucunde*. Small bits of tissue placed in abundant 96 per cent. alcohol, changed twice, can be cut and stained, if need be, within twelve hours.

It is one of the most conspicuous advantages of the alcohol method that material thus hardened can be cut unembedded. This point has already been mentioned. The fact that every variation in technique, every additional step, tends to alter the

end-picture, is reason for subjecting material to the fewest and simplest manipulations possible in preparing microscopic specimens. Anatomic details, manipulative artefacts, post-mortem changes, pathologic alterations—all these are sufficiently difficult to recognise and interpret, and whatever method will simplify the task by reducing the picture, as closely as may be, to an anatomic-pathologic one, is self-evidently the one to be rigorously followed. Such a method is implied in the axiom which has been repeatedly mentioned, and indicates the use of alcohol material cut without embedding for control pictures in cyto-histologic work.

In a number of laboratories, nevertheless, paraffin embedding is a routine procedure for the methylene blue and similar methods. There are but three possible conditions requiring this step: (1) when specially thin sections are desired; (2) when it is necessary to obtain an uninterrupted series; (3) when from the form or nature of the block complete sections are impossible if cut free.

For usual purposes sections of  $10\mu$  or  $8\mu$  are thin enough, and these are readily cut from unembedded blocks. It is only, therefore, when it is desired to have sections under  $8\mu$  in thickness that embedding becomes necessary, as, for example, in embryologic work. For the routine examination of alcohol-hardened cortex, embedding is not only superfluous but contraindicated, except where preparations from free-cut material are used for control, in which case, for obtaining larger or more complete sections, *e.g.*, with an unbroken pia, paraffin or celloidin blocks are also of value.

The quickness and simplicity of alcohol hardening are not its only or, indeed, its chief recommendations. The microscopic picture is the criterion by which its superiority is established. Sublimate and nitric acid as fixatives, with after-hardening in alcohol, give results to a degree comparable, it is true, and may be called for in the treatment of particularly delicate tissue, where the shrinkage produced by alcohol, if applied to the fresh material, would destroy relations; but for the usual cytologic work on the adult cortex, alcohol stands pre-eminent as a hardening medium.

Strangely enough, alcohol has in many laboratories found, even in its specific field, a formidable rival in *Formol*. This



reagent, first used for its antiseptic qualities by LOEW (1888), was introduced comparatively recently into histologic technique by F. BLUM (1893), and acquired a rapid and widespread popularity. By a chemic reaction through which the consistency of the albumens is increased, tissue fixed in formol retains approximately its natural form and colour, the actual weight being somewhat raised, while alcohol causes a progressive loss in weight through the abstraction of water and breaking down of the constituents of the myeline sheaths.

As a *preservative* for gross specimens formol is by all means the ranking medium; moreover, in certain special histologic procedures, such as the demonstration of glia fibres and neurofibrils, as well as in the fixation of mesodermal structures, particularly hæmatogenous elements, formol fills an important place; but it is *not* a reliable *cell fixative* for the ectodermal components of the nervous system, and it is a fundamental error to recommend it, as certain authors do, as a universal fixing and hardening agent for nervous tissue. For the sake of convenience, some anatomists place the fresh material at once into formol or inject the brain with it before removal from the skull, and, after the tissue has acquired a certain consistency, cut out small pieces for cytologic study and transfer them to alcohol. This is also an unfortunate procedure.

The relative value of alcohol and formol as cell fixatives can be established by a very simple experiment. One has but to fix bits of fresh tissue at the same time and under similar circumstances in each of the two fluids, and afterward cut and stain them in like manner with methylene blue or other elective basic dye. There will be found to be no comparison in the results, at least as far as structural details are concerned. The alcohol material will furnish sharp, brilliant "æquivalent-pictures," in which all the nervous elements are fixed alike in approximately their natural form and size, and stand out distinctly against a nearly colourless background, while the formol specimens appear more or less diffusely stained without contrasts, owing to the fact that the ground substance also takes up the dye, and with no satisfactory differentiation of histologic minutiae. In addition, there occurs widespread a peculiar nerve-cell artefact,—artificial shrinking (*künstliche Schrumpfung*, NISSL) which appears to be due directly to the action of formol. Cells thus

affected are shrunken, with concave sides ; display long, slender, often sinuous processes, the capital process particularly assuming a corkscrew appearance, and cell body and processes alike staining so intensely that details of structure cannot be made out. As a result of this change, the nerve cells appear as small, dark, angular bodies in wide gaping pericellular spaces. This phenomenon is pre-eminently characteristic of formol tissue.

The differences above mentioned are the more striking the longer the tissue has remained in formol, which produces in the cell protoplasm a *progressive tinctorial incapacity* ; and although the defects may be inconspicuous to casual observation when the tissue has *very* early been transferred to alcohol, they have nevertheless to be reckoned with from the first. Bearing in mind, moreover, the axiom of celerity, simplicity, and a minimum of manipulation, it is obvious that tissue in which studies on cell protoplasm and nuclei are to be undertaken, and which is therefore to be hardened in alcohol, should be hardened exclusively in alcohol, formol, of all media, not being allowed to come into contact with it. To any one who has made the comparative experiment described, accounts of the "siegeslauf" of formol as a universal fixative are incomprehensible.<sup>1</sup>

As a final fact is to be mentioned that the progressive tinctorial incapacity produced by formol results in the total unfitness of old formal material for histologic purposes, while tissue hardened in alcohol, if thoroughly dehydrated at the beginning and kept so by a sufficiently frequent change of fluid, is satisfactorily available for years, the tinctorial capacity of the tissue being preserved practically intact.

As has been seen, modern technique up to 1890 was chiefly represented by two great methods, the myelinated fibre stain of WEIGERT and the elective cell stain of NISSL. Let us see how much territory was covered by these two methods, and what was left to be demonstrated by other processes. The WEIGERT

<sup>1</sup> POLLACK, in the third edition of his well-known "Färbetechnik" (1905), in discussing the value of formol, makes the following erroneous statement (p. 16) : "Einer der grössten Vorteile aber besteht darin, dass nach Formolhärtung nunmehr *jegliche* bedeutsamere Nervenfärbung ermöglicht ist, sowohl die *Nissl'sche*, wie die *Golgi'sche*," etc.

MALLORY and WRIGHT, in the third edition of their "Pathological Technique" (1904), likewise refer to formol (p. 330) as "likely to find its greatest use histologically as a fixative of the central nervous system."

method, it may be assumed, reveals *a certain part of the course of every axis-cylinder*, understanding by axis-cylinder the group of neurofibrils enclosed in a myeline sheath and continuous centrally with the neurofibrils of the axis-cylinder process of a ganglion cell. To speak of "naked axis-cylinders" is, as BETHE pointed out, nonsense. It is therefore only the true axis-cylinders whose course is made visible by the myelinated fibre method. Naked neurofibrils and neurofibril bundles do not appear. Moreover, it is only in the second of the three divisions of its course that the axis-cylinder can be followed—namely, that portion between the point of constriction of the nerve process at which the neurofibril group becomes invested with the myeline sheath, and the distal point, be it far or near, where the latter suddenly ends, and beyond which the nature, course, and connections of the neurofibrils are unknown. The fibre method is really a macroscopic method. It concerns itself with tracts, fibre systems, cortical fields. Histologically it is of little moment, inasmuch as it throws no light on the most important portion of the course of the neurofibrils, namely, their terminal relations.

The elective cell methods likewise reveal *a certain portion of every cellular element*, both nervous and non-nervous. Of the ganglion cells the general contour and form of the cell-body are indicated, with short proximal portions of the dendrites and the cone of the axis-cylinder process. Within the cell-protoplasm only a certain definite substance or substance-group normally retains the stain—the so-called tigroid or stainable substance of NISSL. The structure of the remainder of the cell body, represented chiefly by the stainless tracts between and through the chromophilic particles, remains entirely invisible. Similarly, in the nerve-cell nucleus it is only the nuclear membrane, nucleolus, and polar bodies, with meagre suggestions of a loose reticulum containing chromophilic granules, which the elective cell method brings to light. The chief mass of the nuclear content is not revealed. In many elements, indeed, particularly the motor cells, the nucleus may appear, except for the nucleolus, almost entirely empty. Nerve cells, in which the stainable substance has reached a certain degree of disintegration, show, by the elective method, a delicate underlying honeycomb structure in the cytoplasm. This structure is exquisitely demonstrated

in the early stages of formation of the golden - yellow pigment.

In the glia cells, as well as in the elements of the vascular walls, other relations obtain between cell-body and nucleus. In these forms it is the nucleus rather than the cell-protoplasm which is conspicuous. The glia nuclei show a more considerable reticulum and granulation than ganglion cell nuclei, while their surrounding protoplasm may be all but invisible. The fibrillar nature of the glia is not revealed, although the course of the glia fibres is in some instances faintly outlined, for example, in the lamina zonalis.

Regarding the essential nature of the intercellular substance, the elective cell method offers no clue.

With all the light which the methods of WEIGERT and NISSL have thrown respectively upon the coarser and finer structure of the central organs, and with the enormous advances which they have made possible both in the normal and morbid anatomy, they have nevertheless left untouched the morphologic units of prime importance, namely, the elementary fibrils both of the non-nervous intercellular substance and of the unstained paths in ganglion cell bodies, and of the axis-cylinders of nerve fibres. These structures remained for other methods to demonstrate.

### (C) *Neuroglia.*

The first of these problems was resolved by the second epoch-making discovery of WEIGERT, that of the elective glia fibre stain, published in 1895. As has been said, the supporting substance was discovered and named and neuroglia fibres recognised by VIRCHOW long before staining or even perfected hardening methods came into use. Not only, however, did the details of the topographic distribution of glia elements remain unknown prior to the studies of WEIGERT, but the following three fundamental points were still involved in obscurity :—

(a) The origin of neuroglia tissue.

(b) The relations between neuroglia and mesodermal connective tissue.

(c) The distinction between and relations of *cell process* and *glia fibre*, as applied to the intercellular supporting substance.

In 1888 WEIGERT began his series of experiments. A method by which glia fibres could be stained and studied was

soon found (preliminary publication, 1890), but in the perfecting of this method, in the restless and soul-trying efforts to make it fulfil *all* the conditions which he had set forth as essentials of an acceptable elective process, throughout all this labour of love stretching over fourteen years, and ending, still incomplete, with the master's death, there runs an undernote of disappointment, of discouragement which at times seemed almost to overshadow the triumph of the brilliant results already attained.

In the first communication (1890) WEIGERT described his method as "beinah fertig." It had indeed yielded a number of fundamental facts regarding the morphologic and topographic relations of the glia, and WEIGERT had concluded with RANVIER upon the absolute independence of nuclei and fibres. The method, however, was incomplete and uncertain, not satisfactorily fulfilling the second of the seven conditions laid down, and in this stage of "beinah fertig" it remained when, after five years more of uninterrupted labour, the now classic work of 1895 appeared. At the close of this work WEIGERT said:—" *Den Abschluss der neuen Methode, den ich jetzt erreicht habe, kann ich nur als einen vorläufigen ansehen. . . . Einer sehr wichtigen Forderung, der der Sicherheit ist im idealen Sinne noch nicht Gütige geleistet. Ehe aber die Methode nicht eine geradezu mathematische Sicherheit besitzt, ist sie nicht als vollendet zu bezeichnen.*"

Finally, in 1902, the long-striven-for result of "mathematic certainty" remained as illusory as ever. In his article on Neuroglia Staining in the "Encyclopedia for Microscopic Technique," WEIGERT declares:—" *Die Hauptsache ist heute ebensowenig bei meiner Methode erreicht wie vor fast sieben Jahren.*" During these years experiments had been going on continuously, and WEIGERT no longer used the method of 1895, as then described. His later modifications, however, he refused to publish, inasmuch as the technique, although improved, was still imperfect. The method was only "beinah fertig," and so it remained when WEIGERT died.

With this method, nevertheless, which for fourteen years was a constant source of anxiety to its author, because of its lack of mathematic certainty, a condition "aus dem sie absolut nicht herauszubringen war," the essential facts in the nature and distribution of the glia fibres were established. It is a method full of surprises and disappointments even for the experienced tech-

nician ; indeed, the results which WEIGERT obtained have scarcely been reached by any one else, and yet a better method we do not possess. Once successful, the pictures it yields are beyond compare, and its complexity and uncertainty are misfortunes, shared by all glia methods hitherto described.

The first question in the development of WEIGERT's technique was that of a mordant. That a mordant was unnecessary for the simple staining of glia fibres was, of course, recognised, and WEIGERT had found that his fibrin method alone was sufficient to reveal fibres here and there. But the conditions he imposed were stricter. In a given area *all* the fibres assumably there present must be brought to light, and must present in addition a maximum of staining intensity and of contrast to surrounding structures. To satisfy these conditions a mordant was required. A metallic salt combined with an organic acid was empirically found to be essential, although the relation between the salt and acid was not clear. In seeking the explanation of this relation WEIGERT finally came to the following theoretic conclusion<sup>1</sup>:—"The stain clings to the neuroglia only when the latter contains a strongly reduced metallic salt. It is impossible, however, for the glia to unite directly with a reduced metallic salt. The union can be effected only with the metal in a highly oxydised state. It is necessary, therefore, first to bring together the neuroglia and the highly oxydised metal, and then to bring about the reduction."

The principle being thus set up according to which the method was to be developed, it was a subject of long experiment to determine the proper salt and acid and their combination. The chrome salts, which are at the same time hardening agent and mordant, were naturally first tried, but the trial led eventually to their abandonment, inasmuch as axis-cylinders also took up the stain along with the glia fibres in tissue thus mordanted. Other hardening media were then resorted to. Formol, then just introduced, was given a careful trial, was found to penetrate the tissue and fix the glia fibres very rapidly, and was adopted as the most favourable fixing and hardening fluid.

During these observations WEIGERT made a discovery of great moment, confirming a previous observation of VIRCHOW. This was the fact of the extreme susceptibility of glia fibres to

<sup>1</sup> Beiträge zur Kenntnis der normalen menschlichen Neuroglia, 1895, p. 184.

post-mortem changes, and of the necessity of bringing material into the fixing fluid *fresh*. This was soon found to be the *conditio sine quâ non* for trustworthy results.

The outcome of the experiments was the discovery of the "typical neuroglia mordant"—the well-known copper acetate—chrome alum—acetic acid mixture. The process was considerably abbreviated later by combining the steps of hardening and mordanting, small pieces of tissue being brought at once into a 10% solution of formol in the copper mixture, and kept in the oven for four or five days. The whole process thus resolved itself into three stages:—

- (a) Hardening—mordanting.
- (b) Reduction.
- (c) Staining.

Blocks from the formol-copper solution were rapidly dehydrated, mounted in celloidin and cut, the sections being then ready for reduction. Here again a great variety of procedures was tried before it was established that the method of LUSTGARTEN, using potassium permanganate and sulphurous acid, gave the most satisfactory results. WEIGERT intensified the reduction process, at the same time securing a slight contrast effect by the addition of chromogen to LUSTGARTEN's solution.

The final process, staining the glia fibres, was accomplished by means of a modification of WEIGERT's fibrin stain, the entire process, including the three stages, requiring twelve days. Later modifications in the first two steps, both by WEIGERT and others, have still further shortened the process, which may now be completed in five or six days.

It was the foundation-laying work of WEIGERT which cleared up the most important facts in the histogenesis and anatomy of the neuroglia. Perhaps its most important contribution was the settlement of the question of the place of mesodermal connective tissue in the central organs. This question was answered in a very direct way by showing by means of the elective stain that the non-nervous tissue which earlier authors (DEITERS, GERLACH) had described as elastic or connective tissue belonged in reality to the true glia, and that mesodermal connective tissue does not enter at all into the make-up of the nervous parenchyma, existing only in the walls of the blood-vessels. Moreover, the glia being

a tissue of single origin,<sup>1</sup> we have the entire nervous substance derived from a single source—the ectoderm, the only mesodermal elements known being those which penetrate from without as vascular mural components or derivations. As a supporting tissue in the sense of the glia, the mesoderm never figures.

Somewhat less fortunate were the deductions of WEIGERT concerning the spatial relations of the glia fibres. His views, agreeing with those of RANVIER, represented a timely reaction to the teaching of the GOLGI school; but the pendulum swung too far. For WEIGERT glia cell “processes” as such existed only in embryonic life. The *appearances* of processes in adult tissue treated by the GOLGI or other non-elective method, were deceptive, and due to the presence of fibres which were in reality chemically and anatomically independent of the cells, and represented, in fact, a differentiated cell protoplasm, but which on account of the inadequate technique, did not present in their true relations. According to this view the adult glia was composed of *cells* (strictly, nuclei) and *fibres*, the two being mutually independent, and inasmuch as the latter constituted the preponderating and obviously more important elements, the term “neuroglia” was reserved for them. WEIGERT admitted the possibility of the existence of an intercellular substance belonging to the glia apart from the fibres, but regarded it only as a possibility. It remained for a later school, among whom especially HELD, to reconcile the teaching of WEIGERT with that of his opponents and show that both sides possessed a part of the truth—that (1) cells, (nuclei and cell bodies with protoplasmic processes), and (2) differentiated fibres, whether anatomically independent or not, and (3) intercellular protoplasmic fibreless glia may all exist together.

#### (D) *Neurofibrils.*

The final task of modern technique was the revelation of the primitive conducting elements which make up the axis-cylinder and occupy the stainless tracts of the nerve cell. The fibrillar structure of nervous elements was established by MAX SCHULTZE,

<sup>1</sup> HIS, one of the first and strongest supporters of the theory of the double origin of the neuroglia, long maintained that DEITER's cells were mesodermal elements. He at length, however, withdrew this opinion, and gave his adherence to the unitarian belief in regard to the source of the glia.



and had indeed been described by REMAK long before, as has been said; but the first actual demonstration of the neurofibrils themselves by means of a staining method was accomplished by KUPFFER in 1883. The method of KUPFFER, hardening in osmic acid and staining in acid fuchsin, applied, however, only to peripheral nerves and myelinated fibres, and is not to be reckoned with the specific tinctorial processes which we have thus far been considering.

Previous to the work of APÁTHY no method had been discovered which was able to bring general conviction among brain anatomists of the individuality of the elementary conducting structures of nervous tissue, including both fibres and ganglion cells. The majority of observers regarded the so-called fibrils as deceptive appearances or artefacts. In the very year in which APÁTHY'S memorable work appeared (1897), CAJAL, describing the spongionoplasm of the ganglion cells in the cord, expressed his belief that the various accounts of a fibrillar structure in the bodies of nerve cells were based upon a misinterpretation of the fine lines of the spongionoplasm which form the delicate ground-mesh in the polygonal spaces of which the NISSL-bodies are lodged.

To APÁTHY is to be ascribed the first unequivocal demonstration of the facts by an absolutely elective process which revealed the neurofibrils "like telegraph wires against a clear sky." The pictures of APÁTHY were indeed so beautiful, so schematic, that his findings were greeted with universal scepticism. The preparations themselves, however, compelled belief. BETHE, in a characteristic passage, thus describes his first acquaintance with APÁTHY'S work. Having read the early published views of APÁTHY, he had communicated to the latter his doubt as to their accuracy. "I had declared to him that I regarded what he had described as lying beyond the range of all possibility, and that his belief that he had followed for millimetres as individuals, fibrils of such fineness, could only rest on self-deception. What was then shown me was so convincingly clear that I was forced, after a few patched-up objections, to withdraw my opposition entirely. As it fared with me, so fared it with many others, and no normal seeing person can escape the convincing impression which the preparations of APÁTHY make, provided that his eye is not dimmed by envy or injured pride."

APÁTHY's specimens, although brilliantly and conclusively demonstrating the nature of the conducting elements in the lower animal forms used for the experiments, were yet subject to the objection that no general application of the method could be made to the vertebrates. Regarding the structure of human tissue, the technique of APÁTHY was not destined to bear evidence.

This rôle fell first to the method of BETHE (1898), with his nitric acid-ammonium molybdate-toluidin blue method. BETHE found that the conclusions of APÁTHY regarding the disposition of the neurofibrils in invertebrates were in all essential points applicable to vertebrates as well.<sup>1</sup> The anatomic individuality of the neurofibril as the fundamental element of the human nervous system was now established. Nevertheless, so great were the technical difficulties of both procedures, the sublimate-gold chloride method of APÁTHY, and the molybdate-toluidin blue method of BETHE, that neither was calculated to occupy a place in everyday laboratory methodique.

In this respect the method of BETHE fared somewhat better than APÁTHY's. The latter has indeed yielded no results in other hands than those of the discoverer himself. So incomparable, however, was the single offspring of APÁTHY's technique, that to the reproach of unfruitfulness it might justly reply with the lioness in the fable: "Ich gebar nur eines, aber einen Löwen!" The method of BETHE, although likewise notoriously fickle, has yet been used with success by numerous observers. It contains, however, the very considerable defect, which was emphasised by WEIGERT in his second postulate, that the tinctorial result is directly dependent upon the regulation of certain steps to minute intervals of time, a few seconds, more or less, prejudicing the success of the stain. The method, like that of APÁTHY, can serve at most no further purpose than that of anatomic demonstration. Its extreme uncertainty and the varying degree of completeness of the fibril pictures it yields, rule it out as a means to histopathologic analysis. S. MEYER's criticism of his own fibril method (iron impregnation) along with BETHE's,

<sup>1</sup> "Im grossen Ganzen verhalten sich die Fibrillen bei Wirbeltieren ganz wie bei wirbellosen, und irgend etwas prinzipiell den Apáthyschen Aufstellungen widersprechen des habe ich nicht gefunden."—*Allgemeine Anatomie u. Physiologie des Nervensystems*, 1903, p. 19.

is very significant. "Both methods would become practically worthless if a fibril method as certain as the NISSL method should be found."

Obviously it was in the field of fibril technique that the most urgent need in modern neuro-histology existed. The closing years of the nineteenth century had supplied a thoroughly reliable cell method, and nerve fibre and neuroglia stains also in a high degree satisfactory, all of which were available for the determination not only of anatomic but also of pathologic details. The ganglion cell as a protoplasmic unit with processes, and containing chromophilic substance, whose axone is continuous with the axis-cylinder of a myelinated nerve fibre; the glia as represented by disseminated cellular elements with conspicuous nucleus, and an extensive system of intercellular fibres—these had become common property. The neurofibrils were more select. Only on the days sacred to the patron saints of histology were they readily conjured into visibility. At this juncture there appeared among the first fruits of twentieth-century technique two methods, both dependent upon the principles of photography, each of which speedily won ardent supporters, and which, in the results they have already yielded in opening up the hardly accessible field of fibril histology, give fair promise of filling up one of the most considerable and most important gaps in our knowledge of the make-up of nervous tissue. These methods were those of CAJAL and BIELSCHOWSKY, both made public in 1903.

The former is far the simpler of the two, but offers less valuable results. The method, as described by CAJAL, consists essentially of but three steps:

(1) Fixation and impregnation of tissue *en bloc* with argentum nitricum.

(2) Reduction of the silver as in the process of developing a photographic plate (acidum pyrogallicum vel hydroquinon).

(3) Final hardening, embedding, sectioning, and mounting.

The simplicity and ease of application of this method, as compared with the complicated technique of BETHE are strikingly apparent. Like the latter, however, it is for the most part useful only for anatomic demonstration. For the study of morbid changes in the fibril system it falls far behind the method of BIELSCHOWSKY. This is an inevitable consequence of CAJAL's *en*

*bloc* manipulations. In a bit of cortex the slowly penetrating silver is densely deposited in the outer layers before effectively reaching the inner zone. One has, therefore, always to be content with a middle zone of varying width between the outer overstained and inner unstained layers. While, therefore, satisfactory pictures may be obtained of the fibril disposition in *isolated cells or cell-layers*, the *vue d'ensemble* of the cortex is an impossibility by CAJAL's method. This defect is a serious one from the anatomic standpoint, but more especially from the pathologic. Here, as is well known, the study of single cells affords no conclusive evidence as to the nature of the morbid process, and it is only by a comprehensive survey of the cortex *vertically* from the pia to the solid white, and *horizontally* over various separated fields, that approximately safe deductions may be drawn.

The CAJAL procedure, further, does not afford ideal contrasts. To this end VAN LENHOSSÉK suggests adding, as a final step, a gold toning bath borrowed from the description of BIELSCHOWSKY, and points out that a comparison of the pictures thus obtained with those of the original method recalls that between a successful PAL preparation of myelinated fibres and an original WEIGERT.

The conditions of an elective fibril method are probably more fully met by the technique of BIELSCHOWSKY than any other at present known. The method depends upon the reducing action of aldehydes (formaldehyde) upon silver in solution (silver-diammonium nitrate). Freezing is preferred to embedding, and the steps of impregnation, reduction, and toning are all accomplished on the cut sections. In this way the unfavourable effect of alcohol upon the silver reduction products in tissue impregnated *en bloc* is avoided, and by treating individual sections much more satisfactory *ensemble* pictures are obtained. VAN LENHOSSÉK, who prefers the CAJAL method as supplemented by a toning process, sees in the freezing technique of BIELSCHOWSKY a serious disadvantage, "in that all nerve cells, particularly the larger ones, are markedly shrunk, and appear surrounded by a strikingly wide artificial space." This appearance is certainly less due to the action of cold than to that of the fluids. Formol has a notorious tendency to the production of this particular artefact. Shrinkage phenomena due to the block-impregnation also appear in CAJAL preparations.

The danger of error in interpreting results obtained with

new fibril methods is illustrated by comparing the findings of CAJAL and VAN GEHUCHTEN on the one hand, with those of HELD and WOLFF on the other, regarding the *epicellular axonal terminations*. These structures ("*Endfüsse*" of HELD; "*Endkeule*" of AUERBACH) are described by CAJAL as terminal enlargements ("*Varicosidades terminales*") of pericellular axones lying upon the surface of the cell, but not uniting with it, and having no relation with its fibrils. Indeed, CAJAL insists that there is always a peripheral zone of cell protoplasm without fibrils, which he misnames the "cell membrane," and which he assumes to represent an uncrossed barrier between pericellular and intracellular neurofibrils. This view, which is also accepted by VAN GEHUCHTEN and VAN LENHOSSÉK, is brought forward in support of the contact theory, and is believed by its adherents to be a conclusive argument against the position of the anti-neuronists.

HELD, who first described these epicellular structures (1897), comes to conclusions entirely at variance with those of CAJAL, VAN GEHUCHTEN, and VAN LENHOSSÉK, although he also made use of CAJAL's technique. He finds that the "end-feet" are not isolated, independent structures, but rather stellate bodies with anastomosing branches, which become intimately united with the ganglion cell in the form of a rich epicellular meshwork, through which run terminal axonal fibrils. These end-fibrils, according to HELD, anastomose with the endocellular fibrils, and the end-feet represent therefore "*Verbindungsstellen zwischen räumlich getrennten Ganglienzellen des Centralnervensystems.*" These results directly oppose the contact theory, and speak for the doctrine of continuity in the sense of APÁTHY. WOLFF, working with the method of BIELSCHOWSKY, agrees with HELD that the end-bodies are not round, homogeneous, circumscribed structures, but in reality fine neurofibril meshes embedded in protoplasm. He likewise believes the appearances obtained by CAJAL and VAN GEHUCHTEN to be artefacts due to imperfect impregnation. BIELSCHOWSKY, speaking of the relations in the cells of PURKINJE, says: "Given PURKINJE cells with distinctly stained neurofibrils, it is scarcely possible to say where the superficial reticulum ceases and the intracellular fibril-mesh begins. In good preparations one gets a definite impression of continuity between the substance of the final axone-structures and that of the cell."

The pathology of the neurofibrils has been attacked from

many sides since the initial work of BETHE, and particularly during the past two years has the field been eagerly worked. Various types of degenerative change have been described, but as yet there is no common agreement as to the occurrence and significance of pathologic alterations. Specific morbid changes in the neurofibrils have not been demonstrated.

The field of the immediate future in neurohistology lies clearly in the realm of fibril relations and connections—a realm to which APÁTHY pointed out the straight and narrow way, so hard to follow, and which may now be approached by the broad highway indicated by CAJAL and BIELSCHOWSKY. The goal of the further extension of this field will be the final clearing up of the nature and office of the peri- or epicellular structures so variously described by different authors, of the *Golginetz*, the *Füllnetz* of BETHE, and eventually, perchance, the elusive *nervöse Grau*.

In the foregoing sketch the attempt has been made to indicate only the most salient points in the life history of neurohistologic technique, extending over the past hundred years. We have seen that the century could be divided into three periods—the first embracing roughly fifty years, during which hardening methods were rudimentary, and staining unknown; the second, covering thirty years, marked by the introduction and domination of the carmine stain, and during which fibre anatomy flourished; the third and shortest period, including the past two decades, during which neurohistology proper has reached its many-sided development through the successive discovery of specific processes for the demonstration of special tissue-components. In the consideration of this final period only the four great methods or method-groups have been discussed, which have been intimately bound up with the discovery of the anatomic details of the several tissue-constituents for which these methods are respectively elective, and which are to-day relied upon in their histopathologic analysis. Other methods have been purposely omitted.

For the myeline sheaths there is practically but one method—that of WEIGERT, which, in some of its modifications, notably those of WOLTERS and PAL, is the one universally employed.

Similarly, in connection with the cellular elements, only the NISSL-technique has been considered. This may, however, be

made to include not only the staining with methylene blue, but also with other basic synthetic dyes, especially toluidin blue, thionin, polychrome, methylene blue, kresyl violet. The process remains the same, merely substituting one stain for another. By this means valuable supplementary or complementary effects may be obtained. For example, thionin and polychrome methylene blue are more metachromatic than toluidin blue and methylene blue, while kresyl violet differs from the others in having a special affinity for nuclear structures, and may thus advantageously be used in association with methylene blue, which has a stronger affinity for the protoplasmic structures of the cell body.

A single glia method has also been described. WEIGERT's method succeeds wherever any method will, and the pictures it yields are unexcelled. HEIDENHAIN's iron-alum hæmatoxyline method is often worth trying. It is extremely simple, and in many instances furnishes surprisingly beautiful and complete pictures of glia fibres, nuclei, protoplasmic cell bodies, and processes and their mutual relations.

In the neurofibril method group, four procedures have been mentioned, representing various stages in the development of this, the youngest branch of neurohistologic technique. APÁTHY's method opened the field with the incontrovertible evidence of the isolated fibrillar conducting paths in invertebrates. BETHE extended the proof to include man. Finally came the simplification of technique with increased certainty of results, through CAJAL, and especially BIELSCHOWSKY, whose impregnation procedure seems called to an important place in the service of neurofibril histology and pathology.

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### ERB'S JUVENILE DYSTROPHY.<sup>1</sup>

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THE four cases reported in this paper belong to a class of affections known as muscular dystrophies. The various dystrophies represent types of one disease and not separate

<sup>1</sup> A paper read before the Leeds and West Riding Medico-Chirurgical Society.

morbid entities. The group includes, besides Erb's juvenile dystrophy, pseudo-hypertrophic paralysis, a dystrophy of the Landouzy-Dejerine type and intermediate forms, such as the Leyden-Moebius and the Zimmering types. The unity of these types is shown by the many peculiarities which they have in common, such as the hereditary and family factor, the occurrence of different types in the same family, the existence of transitional forms, and the tendency shown in the advanced disease towards the equalisation of differences between the various types. Their main differences lie in the primary localisation of the early disease and in the variation in the volume of the affected muscles.

Our knowledge of the dystrophies is of relatively recent origin. As might be expected, they did not escape the keen observation of Duchenne. Leyden drew attention to the hereditary and family factor, and Moebius brought together a couple of types of the disease. To the French school, including Charcot, Landouzy, Déjerine, and others, we owe much useful work in differentiation. Finally, Erb, in describing a hitherto not recognised type, added harmony and completeness to our knowledge of the dystrophies.

The following are short details of four cases of Erb's juvenile dystrophy which came under my observation at the beginning of this year. The family consisted of the father aged 49, mother aged 36, four sons aged respectively 18, 17, 11, and 2 years, and four daughters aged respectively 20, 15, 13 and 7 years. The following members of the family are affected with the disease :—

1. Father. The disease commenced at the age of 19 years. Now the scapulæ are markedly winged. The pectoralis major, serratus magnus, biceps, brachio-radialis, are much wasted. The long flexors of the fingers are also affected. The deltoid and the infra- and supra-spinati are prominent. He has a slightly waddling gait.

2. Son, aged 18. The disease began at the age of 15. He was admitted into the Leeds General Infirmary on February 18th, 1905. The scapulæ are winged. The pectoralis major, latissimus dorsi, serratus magnus, biceps, brachio-radialis, and triceps are wasted. The deltoid and the infra- and supra-spinati are prominent. The levator anguli scapulæ and neck



muscles are unaffected. There is some wasting of the small muscles of the hand. The anterior muscles of the thigh are wasted, especially on the right side. The glutei muscles are prominent. The gait is slightly waddling. The face is unsymmetrical.

3. Daughter, aged 13. The disease commenced about the age of 11 years. She was admitted into the Leeds Infirmary on March 10th, 1905, with pronounced anæmia, and with œdema of the legs. The pectoralis major, serratus magnus, lower trapezius, latissimus dorsi are wasted, and the deltoid is prominent. The biceps, brachio-radialis, and triceps are slightly wasted and weak. She can still swing the arm above the head. The gait is slightly waddling.

4. Son, aged 11 years. The disease began at the age of 8 years. The scapulæ are slightly but distinctly winged. All the movements at the shoulder can be carried out, and only slight weakness is noted.

There is no evidence of any organic spinal cord disease in any of these cases, which show in the main the same clinical features, but in a different stage of development.

The most prominent feature of this progressive dystrophy is the alteration which takes place in the volume of certain muscles, usually in the form of wasting, less commonly of pseudo-hypertrophy. The muscles which are most prone to atrophy are the pectoralis major (especially its costo-sternal portion) and minor, the latissimus dorsi, the lowest trapezius, the serratus magnus, the rhomboids, some of the muscles of the back, the flexors of the forearm on the arm, including the brachio-radialis, the triceps, the quadriceps of the thigh, the adductors, etc. The muscles which are most prone to pseudo-hypertrophy are the deltoid, the infra- and supra-spinati, the gastrocnemius, and some other muscles. The muscles which usually escape are the sterno-mastoid, the levator anguli scapulæ, the muscles of the forearm, hand, and foot. The muscles of mastication and deglutition also escape, even in the advanced disease, so also do the ocular muscles. In Cases 1, 2, and 3 the hypertrophy of the deltoid is very obvious, and the escape of the neck muscles is also illustrated. It is unnecessary to remark that the enlarged muscles show weakness in action, but to a less extent than the atrophied ones. The muscles are

affected symmetrically, but the disease may be more marked on one side than on the other. The direct effect of the muscular disease is deformity, and possibly contracture. The most striking deformity, well illustrated in all of my cases, is due to the displacement of the scapulæ, inasmuch as the primary localisation of the disease, in Erb's juvenile dystrophy, lies in the musculature of the shoulder girdle. The scapula stands at a higher level than in health, the acromial end is rotated downwards and outwards (so that the point of the shoulder becomes lower), whereas the superior angle is higher in consequence of the rotation. When looked at from the front, this angle of the scapula is seen to form a projection on the fold running from the side of the head to the shoulder. Then, again, the vertebral border is more prominent than it should be, and a striking hollow is seen in the space between the two scapulæ. As is to be expected, the deformity is much exaggerated in movements of the arm, especially when the latter is made to assume a vertical position above the head. This movement cannot be voluntarily effected when the disease is at all advanced, owing to the weakness of the lowest trapezius and serratus magnus. Case 3 is only able to bring the arm into this position by literally throwing it up, a fact noticed by several observers of this disease. The scapula is in a singularly loose condition (floating), so that if the hand be placed in the axilla the whole scapula and arm can be lifted up as high as the ear. Another deformity frequently observed is lordosis, and this is well seen in three out of my four cases. It is supposed to be due to weakness of the muscles of the back. Sometimes a lateral curvature is present. The abdomen is abnormally prominent. The gait of these patients is often waddling in character, owing to weakness of the muscles of the pelvic girdle. The pelvis moves laterally on each step.

In three of my cases this peculiar gait is present, and the waddling is more marked on one side than on the other. No contractures are to be seen in my cases.

Another feature in Erb's dystrophy is the absence of fibrillary contractions, such as are seen in muscular atrophies of spinal cord origin. A reaction of degeneration has hardly ever (if ever) been obtained in the affected muscles. As in the other dystrophies, there is an absence of other symptoms often met

with in spinal cord disease, such as bladder and rectal disturbances and alteration in the reflexes (unless the muscles concerned in their production are affected).

A very characteristic feature lies in the distribution of the muscular disease. The proximal part of the limb is the situation in which the disease usually begins. The muscles of the shoulder girdle are chiefly or alone affected in my four cases. The proximal part of the leg is also involved in Case 2, and probably in Case 3. Sometimes the disease begins in the muscles of both shoulder and pelvic girdles. That this progressive dystrophy practically never commences in the distal parts of the limb is a fact of much importance in the differential diagnosis from amyotrophies. Of course, as the disease progresses, other muscles become involved, but there are some muscles, already referred to, which are very rarely affected, and then only in the late disease. In Case 2 there is a slight but quite unusual involvement of the small muscles of the hand, and in Case 1 a curious weakness and wasting of the long flexors of the fingers, but these peculiarities obviously in no way invalidate the diagnosis.

Another most striking feature in the clinical history of Erb's juvenile dystrophy is the hereditary and family proclivities which it shows. It is true that occasionally an isolated and, as it were, sporadic case occurs; but, as Erb says, the disease must have a beginning. Often two, three, or more members of a family may be affected, or there is a clear indirect heredity. In my cases there is a well-marked hereditary and family element (father and three children), but in the case of the father there is no evidence of any such factor; the inquiry, however, has not been by any means exhaustive. Sometimes the disease has been known to skip a generation. Haushalter, in a full and admirable account of the disease, refers to an instance of three dystrophic individuals born of the same healthy mother, but having different fathers. A relative of the mother suffered from the same disease.

The age at which the disease begins is very uniform. It usually appears about the age of puberty, occasionally earlier as in two of my cases, but very rarely after 20 years of age. Case 1 began about the age of 19, and Case 2 at 15. It is thus distinctly a disease of the developmental period of life.

The progress of the disease is very slow and may be interrupted by periods of apparent arrest, but, as Starr puts it, with no attempt at repair. The patient may have the disease for forty years, and perhaps even longer. In Case 1 it has already lasted thirty years. Death, when it occurs, is generally due to intercurrent disease, especially pulmonary affections (tuberculosis, etc.). Very exceptionally the disease extends to the intercostal muscles, and death results from respiratory paralysis. The supervention of bulbar paralysis is unknown in it.

The disease may possibly be mistaken for muscular atrophies due to spinal cord disease, or even it is said for peripheral nerve disease. The main differential features have already been referred to. In progressive dystrophies, as Seiffer briefly puts it, (1) the distribution of the disease is symmetrical; (2) the onset occurs in youth (puberty) or even in childhood; (3) there is the family and hereditary element; (4) the reaction of degeneration is absent in the affected muscles; (5) pseudo-hypertrophy is present; (6) the disease develops more rapidly, and (7) fibrillary contractions are absent. There should be no difficulty in distinguishing a dystrophy beginning in the muscles of the pelvic girdle from congenital dislocation of the hip, but I have recently seen a case in which there was apparently some resemblance between the two conditions. Of course, as in all other diseases, it may be difficult of recognition in the very earliest stages. As regards the other dystrophies the diagnosis is concerned in recognising the type of the disease rather than the disease itself.

Practically nothing is known about the causation of the disease, except the family and hereditary factor. It is interesting to note that in Case 2 the boy was employed in a quarry pushing trucks about, from the age of 12½ to 14 years, viz., during the period in which the disease probably began. Thus there may have been overstrain of the muscles of the shoulder girdle.

The exact pathogeny of the disease is a profound puzzle. Starr gives great prominence to the notion of a restricted vitality in the affected muscles. It is well known that certain tissues and organs have a shorter life than the body as a whole. Thus the dystrophic may have inherited muscles endowed with a shorter term of life than is usually the case. After a certain time they wither and die. Babinski and Onanoff have appealed

to embryonic considerations. Damsch points out that the muscles most often affected in progressive dystrophy are those in which developmental abnormalities are frequently met with, as in the case of the pectorales. Erb inclines to believe in some dynamic alteration in the trophic centres for the muscles in the spinal cord. He appeals to some slight changes which have been thought to exist in the central nervous system by some observers. It may be urged against this view that the disease does not correspond to the root distribution seen in localised spinal cord disease. Neither does the disease correspond to peripheral nerve distribution. It is much more prone to affect physiological muscle groups. A striking illustration is seen in the involvement of the bracio-radialis along with the flexors of the forearm on the arm, as in two of my cases.

I have not had the opportunity of specially examining material from Erb's juvenile dystrophy, therefore I shall pass rapidly by the morbid anatomy. The spinal cord has in the great majority of cases been found quite intact. There is what is usually looked upon as a primary atrophy and sometimes degeneration of the muscle fibres, and a secondary overgrowth of the connective tissue element. Occasionally, actual hypertrophy of the muscular fibres has been noted, but the increase in the volume of the muscle itself is due to a deposit of fat between the fibres.

It is obvious, from what has been already said, that the prognosis of this disease is unfavourable so far as the dystrophy is concerned, although it may not materially shorten life. Patients may still remain useful members of the community, especially if the legs are unaffected. Case 1 was able to do considerable work until weakness in the flexors of the fingers appeared. The earlier in life the disease appears, the worse the prognosis. Some look upon pseudo-hypertrophy as in itself an unfavourable sign. Rapid extension of the disease from one group of muscles to another is a bad sign, and I rather fear a more rapid extension of the disease in Case 2 than is usual. More or less acute cases have been described.

Treatment has, unfortunately, little direct effect upon the disease. Arrest is all that can be hoped for. The patient in whom the dystrophy has already appeared, as well as the remaining healthy members of the family, should naturally live under

the best possible hygienic conditions, and country life is the most suitable for them. In the dystrophic family heavy manual work should be avoided before the age of 20 years. Starr strongly recommends regulated massage, with the view of inducing an arrest in the disease. Moderate gymnastic exercises are probably also of service. Galvanism is recommended by Erb. Tissue extracts (thyroid, thymus, muscle juice) have not been known to exercise any real influence over the disease. Case 2, whilst in the Leeds Infirmary, was systematically massaged and treated with thyroid tabloids. A subjective improvement was alone obtained.

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## Abstracts

### ANATOMY.

**THE SUPERIOR CEREBELLAR PEDUNCLES.** (*Les Pédoncules (276) cérébelleux supérieurs.*) A. VAN GEHUCHTEN, *Névrose*, Vol. vii., Fasc. i., June 1905, p. 29.

THE fibres of which the superior cerebellar peduncles are composed are all ascending. Their cells of origin are situated in the central grey matter of the cerebellum, certainly in the dentate nuclei, probably also too in the roof nuclei. It is certain that the peduncles do not receive fibres from the cerebellar cortex, as no degeneration results in them from superficial cerebellar lesions. Degeneration is found only in the homolateral peduncle after unilateral lesions, *i.e.* no decussation of these fibres takes place within the cerebellum, and no evidence could be obtained of the existence of the descending tract to the ventral columns of the spinal cord, which Cajal has described.

The decussation of the peduncular fibres in the mid-brain is complete, no fibres reach the homolateral nucleus ruber or the thalamus. The most ventral fibres of the peduncle do not cross

the middle line with the rest, but form a small accessory decussation. After crossing the fibres split into ascending and descending branches. The latter can be followed caudalwards through the pons to the oral end of the medulla oblongata. This tract diminishes gradually in size during its caudalward course and approaches the raphé, passing medial to the root fibres of the sixth nerve. Its exact termination could not be determined. The much larger ascending tract passes oralwards among the root fibres of the third nerve, through the red nucleus, in which some of the fibres end and to which numerous collaterals are given off; then lateral to Meynert's bundle, between it and to median fillet till it approaches the lateral side of Vicq d'Azyr's tract. Here the bundle bifurcates, the internal branch passing beneath the third ventricle into the opposite thalamus, and the external bends lateralwards into the lamina medullaris interna thalami and terminates in the median portion of the optic thalamus. A considerable number of fibres also end in the third nerve nucleus of the side opposite to the lesion after their decussation. No fibres could be traced into the internal capsule, or to the lenticular nuclei or cerebral cortex.

The superior cerebellar peduncles do not contain any cerebello-petal fibres from the red nuclei or of other origins. Further, these peduncles contain all the cerebello-fugal fibres; no degenerated fibres were observed in the middle or the inferior peduncles after cerebellar lesions. The existence of the uncrossed cerebello-thalamic tract and of the ventral cerebello-thalamic tract, described by Probst, can be positively denied.

These conclusions were arrived at from the study, by Marchi's methods, of the degenerations resulting from recent lesions of the cerebellum. Destruction of the cerebellar nuclei (roof nucleus and nucleus dentatus) was obtained by passing an instrument into their region through the peduncle of the flocculus. In this way the cerebellar cortex received as little injury as was possible from this method of experimentation. Rabbits were the only animals operated upon. Other structures were occasionally injured in the operation; degeneration was obtained in the vestibulo-spinal tract and the dorsal longitudinal bundle, resulting from lesions in the neighbourhood of Deiter's and Bechterew's nuclei.

Both the older and more recent literature on the subject is concisely reviewed by van Gehuchten, and it is shown that the general tend of opinion has been towards the conclusions he adopts.

GORDON HOLMES.

**ON THE COMPARATIVE ANATOMY OF THE ASSOCIATION  
(277) SYSTEMS OF THE MAMMALIAN BRAIN.** (Zur ver-  
gleichenden Anatomie der Assoziationssysteme des Gehirns  
der Säugethiere. ii. Die Fasciculus longitudinalis inferior.)  
REDLICH, *Arb. a. d. neurol. Institut a. d. Wien. Universität*,  
Bd. x., p. 109.

THE first of this series of papers, that on the Cingulum, has been already reviewed in this journal (vol. ii. p. 448), and the aim and the methods of study of the author are there referred to.

In the present instance the fasciculus longitudinalis inferior has been studied in the brains of representatives of almost all the more important classes of placental mammals, and the research has been, in addition, extended to the brains of the aplacentalia. The Weigert-Pal stain has been exclusively used, and though the results seem as complete as can be expected from the study of normal structures, it must be regretted that the more definite and decisive method of Marchi has not been put to service. This lack leaves us unaware of the direction of conduction and of the origin and end of the tracts.

The inferior longitudinal bundle was recognised by Reil almost a century ago, and later was accurately described in part by Burdach, Arnold, and Meynert. By these and subsequent authors it was regarded as a pure association system connecting the occipital and temporal lobes, and consequently intimately concerned in the psychical association of visual and auditory impressions. Ten years ago, however, Flechsig threw doubt on this interpretation, and described the tract as one of the projection systems, its fibres connecting the occipital cortex with the thalamus. Since then this view has been confirmed by the majority of those who have investigated the subject, including Probst, Henschen, Hösel, and Niessl-Mayendorf. Redlich's industrious work has led him to support Flechsig's view in its entirety.

The size and shape of the tract varies in different classes of mammals. It is more complicated in the higher species, in association with the better development of the cerebral cortex, especially of the visual centre, than it is in the lower. In man its cortical distribution is to the medial surface of the occipital lobe in the region of the calcarine fissure, to the basal and convex surfaces of the same lobe, and to the neighbouring parietal gyri. The first definite statement which can be made is that no large bundles enter the temporal lobe from it, though isolated fibres may do so in small number. But it seems probable that fibres leave the tract during its forward course in the lateral wall of the ventricle. When it reaches the level of the thalamus, bundles of fibres turn



medialwards through the retrolenticular part of the internal capsule; those of the dorsal part of the bundle enter the lamina medullaris thalami, while many of the more ventral bundles reach the corpus geniculatum laterale. In addition to these, a few fibres pass forwards into the external capsule. The fasciculus longitudinalis inferior is thus quite analogous to the projection systems of other parts of the brain. This is certainly true for its dorsal part. Its most ventral fibres, however, enter the hippocampal gyrus by passing ventral to the posterior turn of the ventricle, and extend forwards into the uncus and nucleus amygdalus. It seems probable that neither do these fibres comprise an association tract between the occipital and temporal lobes, but that they are projection fibres from the latter to the subcortical ganglia which run in the fasciculus longitudinalis inferior.

The inferior longitudinal bundle forms the lateral part of the stratum sagittale occipitale, and is distinguishable from the median part, the so-called optic radiations, by the larger calibre of its fibres, and by the earlier date of its myelinisation; and in the lower mammals a palely staining zone of doubtful significance separates them. The relation between the two cannot be yet accurately defined. The fact that the fasciculus longitudinalis inferior is earlier myelinated than the stratum mediale suggests that it is corticopetal, and this conclusion is adopted by Hösel and Niessl-Mayendorf, and supported by the experimental work of Probst, and lately accepted by Henschen. The optic radiations of Gratiolet, or stratum sagittale mediale, is regarded by these authors as cortifugal. The method of study employed by Redlich does not allow him to decide the question.

By these conclusions the importance of the association systems—*i.e.* long tracts directly connecting widely separated sensory centres—is further limited, and even their existence becomes questionable.

GORDON HOLMES.

**ON THE GENESIS OF THE CENTRAL NERVE FIBRES AND**  
(278) **THEIR RELATION TO THE GANGLION CELLS.** (*Su la genesi delle fibre nervose centrali e il loro rapporto con le cellule ganglionari.*) FRAGNITO, *Annali di Neurol.*, Fasc. 1-2, 1905.

THE author, who has in earlier papers maintained the pluricellular origin of nerve cells by the metamorphosis of the syncytia, and also the pluricellular origin of the peripheral nerve fibres by the transformation of a series of cells united in a chain, here gives a description of the genesis of the central nerve fibres.

By a slight modification of the fifth method of Donaggio for

staining the intracellular nerve fibrils, he has succeeded in obtaining a clearer demonstration of the chain of cells with their nuclei, and he has been able to follow the disappearance of the nuclei and the formation of the nerve fibrils.

In fig. 1, accompanying the paper, there is reproduced the anterior commissure of the spinal cord of the eighteen-day embryo of a chick. The fibres resemble a ribbon or thread with fusiform swellings at short and regular intervals. With a higher magnification it is seen that the swellings contain nuclear substances.

It is inferred that each swelling represents a cell, and that the whole thread is composed of a large number of cells, whose nucleus is quite evident, and whose protoplasm is elongated into two filaments which unite with the filaments of the two contiguous cells. The author figures one thread in which there are seventeen cells, and he has seen another which contained twenty-two cells.

But the nucleus does not remain very long. It tends to gradually disappear, and probably its substances are diffused into the protoplasm—transformed into the axis-cylinder.

In the cells of the same thread the nuclei are seen in various phases of transformation, and as they fade the fusiform swellings disappear, and the margins of the thread tend to become parallel. This gradual disappearance of the nuclei justifies two hypotheses: either that the nuclear substances, having fulfilled their function, disappear, absorbed by the protoplasm of the axis-cylinder, or they survive in the protoplasm.

According to the author the axis-cylinder is derived from both the substances, nucleus and protoplasm, as suggested by Paladino in 1892.

One important fact observed in the study of these cell chains is that the axis-cylinders in the spinal cord are formed at a relatively late period. La Pegna agrees with Fragnito that the axis-cylinder is never seen in connection with the nerve cell before the tenth day, although there is an abundant formation of dendrites, in which the fibrils are quite in evidence.

This differs from the statement of Ramon y Cajal, that by the fifth day almost all the axis-cylinders have reached their destination, emerging from the cord by the anterior roots as well formed tubes of white matter. Now, leaving out of the question the formations of the first five days, which are neuroglial in origin, Fragnito's investigations show that the dendrites are developed earlier than the axis-cylinders.

In another figure the author exhibits an axis-cylinder which maintains its individuality while passing through the cell-body towards a protoplasmic process. In its endocellular portion the axone presents nuclei arranged as in its extracellular portion.

Also filaments can be seen passing from the perinuclear region to anastomose with it.

Having thus demonstrated the continuation of the chain of cells from which the axis-cylinder is derived, through the body of the nerve cell, and to a protoplasmic process on the other side, the author has filled the important lacuna which was left in his earlier works on the development of the nerve cell.

From these series of cells the neuro-fibrils are in all probability derived. It is also likely that in the nerve cells the arrangement of the fibrils which pass from the protoplasmic processes in compact bundles to the nuclear region, and of the fibrils which start from the nucleus and pass out of the cell as the axis-cylinder, is based on the embryological fact that the chains, forming in their course the cellular prolongations, preserve their individuality when they enter to form part of the syncytium, which is the precursor of the nerve cell. As to the physiological significance of the relatively early development of the connections between the perinuclear zone and the protoplasmic prolongations, the author is not yet able to speak definitely. R. G. Rows.

#### LATER RESEARCHES ON THE GENESIS OF NERVE CELLS.

(279) (*Ulteriori ricerche sulla genesi delle cellule nervose.*)  
F. CAPOBIANCO, *Annali di nevrol.*, Fasc. 1, 2, 1905.

ALTHOUGH the pluricellular origin of the nerve cell is not yet universally accepted, it is being supported by the work of an increasing number of investigators, who have observed it in almost all classes of vertebrates. The author here gives the results which he has obtained by a careful observation of the development of the nerve cells in the cat.

In this animal he has found that the "syncytium" is best seen in embryos of 3-5 cm. length, measured from the vertex to the beginning of the tail, the neck being extended; in man in the embryo of the third month of gestation. It must be remembered that the cells of the various parts of the nervous system do not all exhibit the same stage of development at the same time. The author has observed that from the earliest stages, even in the cat of 2 cm., there is a recognisable zone of protoplasm around the nuclei of the elements which form the syncytia or colonies; this becomes more evident as development progresses.

By means of the excellent series of illustrations which accompany the paper, the author shows the various stages through which the neuroblasts pass. In the first illustration we see the separate neuroblasts, still separate, but approaching each other;

then in a section taken from an embryo 30 mm. long, among the neuroblasts scattered diffusely, are seen others which have approached each other, and which begin to have the appearance of a colony. This is better seen in a section taken from an embryo 33 mm. long.

The neuroblasts have large vesicular nuclei, which lie so close together that the space between them can only be detected by a high magnification. Comparing this figure with the preceding, one sees that it illustrates a phase immediately following the other.

Next there is a section taken from the embryo 43 mm. in length. In this the nuclei are intimately connected, but have not yet fused.

The appearance is that of a neuroblast with two nuclei, one of which is paler than the other, *i.e.* it is undergoing the process of extinction.

This pallor is, without doubt, an index of a chemical degradation which precedes the histological disintegration, because the diminished power of retaining the stain must depend on an altered condition of the chromophile elements. This degradation of the nucleus proceeds gradually until all trace of it is lost.

Such involution of the nuclei must be of the greatest importance in the formation of other elements more complex and more evolved, and the products of the demolition of the former must serve for the construction of the latter, which remain to form the definite anatomical basis of nervous function.

A demonstration of the same process is shown in another section, which is taken from a human foetus of about three months.

As an explanation of these movements of the neuroblasts among themselves, the author suggests that the phenomenon of chemiotrophism must be an important factor in the production of such evolutive processes as the migration of the neuroblasts from the region of the neural canal, and the migration of the mesodermic elements to form the mesoglia.

The complexity of the syncytia differs very greatly, varying with the different types of cells met with in the ganglia, the smaller cells being the products of the simpler syncytia.

In some of the figures shown one can easily also see demonstrated the pluricellular origin of the processes of the cell.

In the development of the cells of the grey matter of the spinal cord the same processes can be followed, but there are slight differences of time. As a matter of fact, all these processes occupy a very small amount of time.

The author next gives an account of a laborious counting of the neuroblasts in embryos of 20 mm., 43 mm. in length, in the new born

and one year old animals, and compares the totals with the number of nerve cells found in the adult animal.

If the nerve cells are formed by the fusion of several neuroblasts, their number must be less than that of the elements from which they are derived. If this could be proved it would decide the question of the pluricellular origin of the nerve cells, as Bethe admits.

In one embryonal ganglion the author found 27,374 elements, and in one adult ganglion 9702 nerve cells, and in another 8538.

The relation between the neuroblasts and adult nerve cells was therefore in the one case 2·83 : 1, and in the other, 3·20 : 1.

R. G. Rows.

**ON THE GENESIS AND RECIPROCAL RELATIONS OF THE  
(280) NERVOUS ELEMENTS IN THE SPINAL CORD OF THE  
CHICK.** (Su la genesi ed i rapporti reciproci degli elementi  
nervosi nel midollo spinale di pollo.) EUGENIO LA PEGNA,  
*Ann. di Neurolog.*, 1904, F. 6.

THE author has used chiefly the new silver method of Ramon y Cajal, but also Donaggio's recent methods for the neurofibrils. His observations support the view of the independent origin of the nerve fibres and nerve cells, and also confirm the opinion of Fragnito and others that the intra-cellular neurofibrils are formed at a comparatively late stage. He states his conclusions as follows. The nerve cell does not take part in the formation of the nerve fibre; the nerve fibre in the first stages of its development has no connection with the nerve cell; the peripheral and central nerve fibres are developed from cellular chains; these cellular chains form only the axis-cylinder of the nerve fibre and do not contribute to the formation of its other elements; the protoplasmic processes, like the axis-cylinder process, are developed from cellular chains; the neuro-fibrils of the nerve cells are a late product of differentiation; in the chick they do not develop before the tenth day of incubation.

W. FORD ROBERTSON.

**THE FINE HISTOLOGY OF THE GIANT NERVE CELLS OF THE  
(281) HUMAN CEREBRAL CORTEX.** (Fina Istologia della Cellule  
nervose giganti della corteccia cerebrale umana.) ROSSI,  
*Nervaze*, Vol. vii., F. 1, June 1905.

IN the protoplasmic processes of the pyramidal nerve cells the fibrils are placed parallel to each other, and joined by fine

trabeculae. The neurofibrils are directed towards the cell centre, where they are lost in the perinuclear network. The cone of origin of the axis-cylinder and protoplasmic processes results from a number of fine fibres proceeding from all directions of the cell.

Fibrils are seen passing from one protoplasmic process to another, and sometimes a fibril of a large protoplasmic process bifurcates at the point of separation of two processes to send a fibril into each. Sometimes between two protoplasmic trunks there exists a true reticulum formed by many anastomoses between the fibrils of each.

There are giant cells in the cortex which, although pyramidal, differ from the preceding in having a more reticular structure.

These have two varieties of network, one of elongated and wide meshes at the periphery of the cell; another more dense, of polygonal meshes, around the nucleus—perinuclear. The primary filaments lie towards the cell periphery, and in the protoplasmic processes this peripheral disposition is well seen. Some sinuous fibrils of unequal size form a network, which gradually contracts towards the nucleus. All the primary filaments are more strongly coloured than the secondary ones, and both are united by fine anastomotic branches. The peripheral fibrils of the axis-cylinder are derived from adjacent protoplasmic processes, and the central from the peripheral and central protoplasmic network.

The giant cell of fusiform shape contains fibrils collected in fasciculi, crowded together in the cell centre, but running into the protoplasmic process in three distinct bundles.

Here again the secondary filaments are less stained than the primary. The cell network is formed of polygonal meshes, with which it is difficult to perceive direct connection of the filaments of the protoplasmic processes. It is also difficult to ascertain the presence of fine anastomotic trabeculae. The author is certain, however, that the cytoplasmic reticulum is not independent of the fibrillar bundles.

The giant oval cells show polygonal meshes. The fibrils of the protoplasmic processes anastomose with each other, and in the cell body are resolved into a very fine network.

*Conclusions.*—In all giant cells fine fibrils form a network whose meshes may be quadrate, triangular, irregularly rectangular, and elongated. More generally they are polygonal.

There are two types of giant cell—one reticular, one fibroreticular. Where the meshes are close together it is difficult to perceive the fine interfibrillar anastomoses, but they are always present in cell body and processes.

DAVID ORR.

**NEW METHODS OF STAINING THE NERVE CELLS (REGARD-  
(282) ING THE QUESTION OF LYMPH CHANNELS). [Neue  
Methoden zur Färbungen der Nervenzellen (zu der Frage von  
Saftcanälchen).] PASSEK, *Neurol. Centralb.*, No. 13, 14, 1905.**

**METHOD I.** Pieces of nervous tissue  $\frac{1}{2}$  cm. in thickness are placed in 15 c.cm. of the following solution:—1 gr. osmic acid dissolved in 100 c.cm. saturated sublimate (without salt)=5 c.cm.; 5.5% watery acetic acid=10 c.cm. Leave the tissue in this for 5 to 7 hours; freeze in chlorethyl, damp the knife with cedar or bergamot oil, pass sections into either oil, then into pure acetone or 95% alcohol for 3 to 4 minutes to remove the oil; then into acetone 80% or alcohol 90% with tinct. iodi for 5 minutes to remove the mercury; then acetone 30% or alcohol 30% for 2 minutes to remove the mercuric iodate; acetone 20% for 2 minutes, then distilled water.

At the periphery of the tissue the nerve cells are intensely stained, but can be cleared up with tannin or Kolossow's developer.

The cells are now stained with hæmatoxylin (Gren). The protoplasm stains grey violet against a black (osmic acid) groundwork of protoplasmic structure and nucleolus.

The nucleolus is surrounded by an unstained canal, from which branch others having distinct walls. Outside the cell there are larger canals all through the grey matter.

**Method II.** Pieces of nervous tissue from  $\frac{1}{2}$  to  $\frac{3}{4}$  cm. in thickness placed for 24 to 36 hours in 20 c.cm. of the following mixture:—1 gr. osmic acid in 100 c.cm. saturated watery sublimate=10 c.cm.; Müller's fluid=10 c.cm. Wash in many changes of water for 24 hours; then in distilled water. Pass through alcohol and iodine, or better, through graduated acetones, beginning at 20%, and in the course of 36 to 48 hours increasing 10% every 3 to 4 hours to pure acetone. Place in cedar oil in thermostat at 37° for 24 to 36 hours; then 36 to 48 hours in the following:—spermacetæ, 40; yellow wax, 1.5; ol. ricin., 10; for 24 hours in thermostat at 45° to 48°.

Colour the sections with Heidenhain's hæmatoxylin or Weigert's resorcin-fuchsin method, with previous staining in 1% iron—sulphuric—ammonia solution. In the latter method (Weigert) differentiate with hydrochloric acid and spirit.

Certain granules in the cells are fixed by this method, and are found scattered in the protoplasm and in the canals. They are not similar to the neurosomes of Heldá, but are perhaps the products of regressive changes in the chromophile elements.

In nerve cells there are three kinds of canals:—

1. A network of arterial branches, which penetrate the cell. These have a wall, which is gradually lost, and merge into drains, which possess no wall, and contain the nutrient lymph of the blood.

2. Channels without walls, to convey the lymph out of the cell.
3. Channels formed by the disintegration of the chromophile elements (not true canals).

DAVID ORR.

**THE SENSORY AND MOTOR NUCLEI OF THE VAGUS IN THE**  
 (283) **RABBIT.** (*Les noyaux sensibles et moteur du nerf vague chez de lapin.*) ALFEWSKY, *Le Neuraxe*, June 1905, p. 23.

THIS paper comes from the laboratory of Van Gehuchten, and the work is a continuation of his investigations into the motor and sensory origins of the vagus in the rabbit. The pharyngeal, superior and inferior laryngeal, and cardiac branches of the vagus were divided, and the nerve cells giving origin to their fibres located by the method of chromatolysis.

The motor fibres of the *pharyngeal branch* have their origin in cells situated in the upper fourth of the nucleus ambiguus, or anterior motor nucleus of the vagus, of the same side, and there alone. The sensory fibres come from cells in the ganglion of the root (g. jugulare).

In the case of the *superior laryngeal branch* the motor fibres come from the upper part of the dorsal nucleus of the same side, and the sensory fibres entirely from the ganglion of the trunk (g. nodosum).

The *inferior laryngeal branch* is purely motor; its cells of origin lie in the lower half of the dorsal nucleus of the same side.

The *cardiac branch* contains only sensory fibres, and these come from the ganglion of the trunk.

Thus, the dorsal nucleus of the vagus innervates the muscles of the larynx; the ganglion of the trunk gives origin to the sensory fibres of the larynx and to all the cardiac fibres; the pharynx receives its motor fibres from the nucleus ambiguus, whilst its sensory fibres come from the ganglion of the root. None of the fibres of the vagus cross in the medulla oblongata; they are all direct, and come from the nuclei of the same side.

SUTHERLAND SIMPSON.

## PHYSIOLOGY.

**THE RÔLE OF CENTRIPETAL AND CENTRIFUGAL STIMULI**  
 (284) **WITH REGARD TO THE FUNCTION AND NUTRITION**  
**OF NERVE CELLS.** (*Du rôle des excitations centripètes et centrifuges dans le fonctionnement et la nutrition des cellules nerveuses.*) MARINESCO, *Rev. Neurol.*, No. 13, 1905.

THE law of Waller, that when a nerve bundle is interrupted so as to hinder its reparation, the peripheral portion, separated from its



trophic centre, degenerates, while its central end, still in relation with its centre, remains normal, has been shown to be fallacious as far as it regards the central stump. Various observers have shown that the central end may degenerate and that the cells of origin may atrophy.

Moreover, this degeneration of the central end is not retrograde or ascending, but centrifugal or descending, a fact which is demonstrated by finding the neurofibrils profoundly altered in the cell and still persisting in the prolongations.

To this condition van Gehuchten has given the name "indirect Wallerian degeneration"—secondary descending degeneration.

But besides this degeneration, Gudden and others have shown that after enucleation of the eyeballs there is an ascending degeneration of the optic system; there is found sclerosis of the optic nerves, slight atrophy of the anterior quadrigeminal bodies, and marked atrophy of the external geniculate bodies.

From a study of lesions following amputations, and of those of the brain after infantile hemiplegias, Marinesco has come to the conclusion that they do not agree with the law of Waller; in the former case there is an atrophy of the white and grey matter of the cord on the same side as the lesion. To explain this he has suggested that the trophic action of the cells of the anterior cornua and of the spinal ganglia is maintained by the normal and regular influx of stimuli from the periphery; in fact, as Goldscheider has said, "the continuity of excitation in all the nerve paths is the basis of trophic action."

Moreover, the second system of neurones has often been found atrophied, and to this the name "secondary neurone atrophy" has been given.

In the cerebellum also, after infantile hemiplegias, there is a great atrophy of the lamellæ, and most of the cells of Purkinje have atrophied or degenerated.

The changes seen are of the primary and not of the secondary type, and are produced by the absence or insufficiency of functional excitations, and not by solution of continuity of the axis-cylinders. These facts prove a close relation between the cells, and if the function of one cell is disturbed for a long time an injurious effect is produced on the nutrition and on the function of the following neurone. In order to investigate the changes produced in the cells when the peripheral stimuli are withdrawn and the cell is so placed that it can no longer functionate, Marinesco has made a transverse section of the cord and has also cut the sciatic nerve.

After three days in the rabbit he found swelling of the cell, the nucleus, and the nucleolus, and chromatolysis or achromatosis of the elements at the periphery of the cell. In the processes the elements were more resistant. After a little time the cell began

to diminish in volume, and the elements began to be reformed in the periphery.

But the repair of the elements did not proceed very far, and most of the cells continued to atrophy and finally disappeared. Some, however, remained in a partially recovered condition.

In the dog the lesions are less severe, and after ninety to a hundred days most of the cells have recovered to a considerable extent. But if the joining of the cut nerve is prevented all the nerve cells atrophy.

In the normal state the nerve cell receives stimuli from the superior centres and especially from the cortex. It also, by the collaterals of the posterior root fibres, receives stimuli from the sensitive surfaces. The motor cell also sends the elaboration of its internal work to the muscles, translating it into tonus.

Now, section of the cord and of the peripheral nerve removes the central and the peripheral stimuli, and abolishes the transmission of the internal work of the cell. The nutritive equilibrium of the cell is disturbed and the conditions mentioned above are produced, conditions quite peculiar, and independent of the reaction caused by the section of the axis-cylinder. As an explanation, Marinesco suggests that it is not a reaction following section of the axis-cylinder, precipitated by section of the cord, but it is due primarily to the section of the cord, which cuts off the stimuli which normally maintain the nutritive equilibrium. In fact the changes seen after three days are primary, and precede the secondary reaction.

Section of the cord and a nerve, therefore, produce a much more serious lesion than simple section of a nerve.

The bundle which conducts these important central stimuli is the cortico-spinal. It is these central stimuli which are necessary for the reparation of the cells.

We may say, therefore, that a cell cut off from the higher centres and from the periphery atrophies. R. G. Rows.

**ON RECIPROCAL INNERVATION OF ANTAGONISTIC  
(285) MUSCLES.** Seventh and Eighth Notes. C. S. SHERRINGTON,  
*Proc. Roy. Soc.* Vol. B. 76, 1905.

THE seventh note deals with the phenomenon of "spinal induction." A reflex arc during its activity temporarily checks the discharge action of an opposed reflex arc, but as a subsequent result induces in it a phase of greater excitability and predisposes the spinal organ for a second reflex opposite in character to its own in immediate succession to itself. The significance of this in the act of locomotion is obvious.

The eighth note deals with inhibition occurring in instances of "reciprocal innervation" obtained as a spinal reflex reaction.

1. Reflex inhibition (relaxation) and reflex excitation (contraction) are part and parcel of one and the same reflex reaction; opposite in direction, they are co-ordinate reciprocal factors in one united response.

2. A reaction which throws one set of muscles into active contraction inhibits the tonus of the antagonistic set, and can also cut short their active contraction.

3. Skin nerves have a reflex inhibitory action, and the arcs arising in the muscles which those cutaneous arcs excite are synergic.

4. The knee-jerk—though perhaps not a true reflex—is dependent on the integrity of a reflex spinal arc which arises in and returns to end in the extensors of the knee; other reflex arcs are inessential to the phenomenon.

5. The process of inhibition—whatever it may be—converse in effect to that which "excites" a contraction reflex, yet so resembles that in time-relation of onset as to suggest that the induction of the state of change in the centre, if active in the one case, is active—not passive—in the other.

Individual afferent nerve fibres from the receptive field of a reflex each divide in the cord into end branches (*i.e.* collaterals), one set of which, when the nerve fibre is active, produces excitation in certain efferent neurones, while another produces inhibition of the efferent neurones of the antagonistic muscles.

Strychnine and tetanus-toxin have the effect of converting the inhibition into excitation, so that protagonist and antagonist muscles are thrown into synchronous contraction.

J. H. HARVEY PIRIE.

## PATHOLOGY.

### THE DEGENERATION AND REGENERATION OF PERIPHERAL

(286) **NERVE AFTER A LESION OF THE SAME.** (*Die Degeneration und Regeneration des peripheren Nerven nach Läsion desselben.*) Dr GUSTAV MODENA, *Arch. aus d. neurolog. Instit. in Wien*, 1905, p. 243.

THE value of the work contained in this paper is to a considerable extent limited to two points—firstly, the nature of the axis-cylinder in peripheral nerves; and secondly, the degenerative and regenerative changes which occur in the axis-cylinder after division from its trophic cell centre. The conclusions at the end of the paper are:—

(1) The axis-cylinders of peripheral nerves are made up of

continuous fibrillæ and a peri-fibrillar substance called axoplasm, which is interrupted at the nodes of Ranvier.

(2) The author has confirmed the statements made by Von Kaplan, Chilesotti, and others, with regard to the colouring of a part of the axoplasm.

(3) The sheath of Schwann is interrupted by the nodes of Ranvier.

(4) The appearances of degeneration begin at the lesion, and extend towards the periphery of the nerve, depending a good deal on the kind of injury to which the nerve has been exposed. In the central end of the divided nerve the changes cease at a short distance above the lesion, provided careful antiseptic precautions have been taken.

(5) The axis-cylinder degenerates in every case, and throughout the whole length of the peripheral portion of the nerve. The fibrillæ and the peri-fibrillar substances undergo a typical granular degeneration. Vacuoles occur, and the whole axis-cylinder breaks up into fragments.

(6) This degeneration of the axis-cylinder only occurs in a few of the fibres in the central end of the divided nerve, and ceases at a short distance above the lesion.

(7) The first changes in the myelin begin at the same time as the changes in the axis-cylinder.

(8) The nuclei of the neurilemma sheath show in 24 to 36 hours after the operation kariokinetic figures, and become arranged in rows, which eventually form new axis-cylinders, and by the Golgi-Cox method of staining the fibrillæ can be seen clearly in 10 to 15 days in the protoplasm which is to form the new axis-cylinders.

(9) After 18 to 28 days the first appearance of colour reaction appears in the axoplasm, forming thread-like appearances in the neighbourhood of the nuclei of the neurilemma sheath, which are, the author believes, neuroblasts.

(10) Soon after the regenerative processes have reached this state the myelin sheath begins to form.

(11) The new myelin sheath is perhaps formed in part out of the pre-existing sheaths, which have undergone destruction, but by the agency of the neuroblasts.

(12) The sheath of Schwann and the neurilemma sheath are closely related in origin to connective tissue.

(13) An adult animal does not show the same amount of regeneration unless after the central ends of the divided nerve become joined to the peripheral end; and peripheral regeneration is best marked in young animals, but it never reaches an advanced stage unless under the influence of the cell centres.

(14) The influence of the cell centres on the peripheral ends

of the divided nerve not merely brings about regeneration in its final stages, but governs entirely the use to which the cell fibres so regenerated can be adapted.

The fixing and staining methods adopted were largely Flemming's solution, osmic acid, weak solutions of sublimate, and the Golgi-Cox method for staining the axis-cylinders. The animals on which the experiments were carried out were rabbits, guinea-pigs, and dogs, and as a rule young animals were preferred. Different kinds of operations were performed, cutting the nerve, removing a part of the nerve with and without interfering with the divided ends, compression with clamp forceps, ligature with horse hair or thin catgut, and in certain cases cauterisation with the Paquelin thermocautery. The utmost care was taken to keep the wounds aseptic, and much trouble was experienced in the case of the rabbits, in many of whom ulcers and joint suppurations occurred on the affected limb. In all the animals the sciatic nerve was chosen. Some of the specimens were examined in a matter of 6 to 36 hours, and the longest period was 180 days after the operation on the nerve. The results at which Dr Modena has arrived have been classified above, and they have been formulated after a careful and prolonged piece of work. Very brief account, however, is given of the details of any particular series of the experiments, the paper being rather a summary of what the author has noted, with a full description of the different staining methods applicable to the investigation of the axis-cylinder and its changes after section. Several well-drawn and coloured illustrations complete an abstract of an excellent piece of work.

ROBERT A. FLEMING.

**COMBINED SCLEROSIS IN GENERAL PARALYSIS.** (*Les*  
(287) *scléroses combinées médullaires des paralytiques généraux.*)  
VIGOUROUX et LAIGNEL-LAVASTINE, *Nouv. Icon. de la Salpêtr.*,  
May-June 1905.

In a large series of cases these authors have observed rarefaction of the pyramidal fibres following diffuse cerebral lesions; mass degeneration of the pyramidal fibres secondary to a focal lesion in the brain or a subdural hæmorrhage; cases in which the lesion corresponded to the primary lateral sclerosis of Westphal, but was really secondary (according to the authors) to myelitis, meningo-myelitis, or lesions of the cord nerve-cells.

They have also observed sclerosis of the posterior columns occurring in some cases of old standing, and unaccompanied by any other medullary lesion, but in others with meningitis and inflammation of the posterior part of the lateral columns. Again,

some cases showed meningo-myelitis and descending sclerosis of the pyramidal tracts from cerebral softening.

Rapid lesions appear in the crossed pyramidal tracts and posterior columns as a result of meningo-myelo-encephalitis; and they have met lesions of the lateral basis bundles due to polio-myelitis, and posterior column sclerosis due to a root lesion.

The authors lay stress upon the great diversities of aspect of the sclerosis in general paralysis and their many exciting causes. These, however, are only evolutive processes in one and the same disease.

DAVID ORR.

### CLINICAL NEUROLOGY.

**A CASE OF SPINAL HEMIPLEGIA WITH RADICULAR DISTRIBUTION IN THE ARM AND CROSSED THERMOANÆSTHESIA — THE RESULT OF HÆMATOMYELIA.**  
(*Hématomyélie ayant déterminé une hémiplégie spinale dans le membre supérieur avec thermoanesthésie croisée, etc.*)  
RAYMOND and GUILLAIN, *Rev. Neurolog.*, July 30, 1905, p. 697.

A YOUTH of 21, when taking a cold bath in the month of August 1904, was suddenly seized with complete paralysis of arms and legs. After some months the weakness on the left side improved, but a Brown Sequard syndrome persisted, *i.e.* right spinal hemiplegia with left thermoanæsthesia.

On examination in June 1905 the flexors of the right lower limb were much more affected than the extensors, and the abductors of the thigh more than the adductors. The flexors of the right arm were in a state of contracture; the last three fingers of the hand were pressed into the palm, but the movements of thumb and forefinger were unimpaired. Further motor difficulties were noted in the distribution of the inferior division of the brachial plexus. Only the triceps showed atrophy and gave the reaction of degeneration.

All that remained of the paralysis in the left leg was slight weakness of the hamstrings; in the arm, slight contracture of the flexors, slight paresis in the muscles supplied by the inferior division of the brachial plexus, paresis of the triceps with the reaction of degeneration. There was no disturbance of sensation on the right side; on the left there was loss of temperature sense in leg and trunk, and in the arm over the areas supplied by the eighth cervical and first dorsal roots. The tendon reflexes of the lower extremities were greatly exaggerated; there was a double extensor response; the wrist reflexes were increased and the triceps jerks abolished.

It was evident in the first place that the hæmorrhage had damaged the segment in which are the cells of origin of the triceps, i.e. the segment corresponding to the sixth or seventh cervical root. The paralysis and contracture of the right flexors, in the absence of any atrophy, indicated a lesion of the white, not of the grey, matter at this level. Interference with the pyramidal path on the left side was clearly very slight.

The point of interest in the case is the fact that the upper division of the brachial plexus is entirely unaffected, and the authors therefore incline to the theory, formulated by Dejerine and Gauckler, that the terminations of the pyramidal path follow in the cord a root distribution.

S. A. K. WILSON.

**TWO CASES OF MENINGEAL HÆMORRHAGE—CONSIDERATIONS ON THE CLINICAL VARIABILITY OF THE SYNDROME.** (Deux cas d'hémorragies méningées: Considérations sur la variabilité clinique du syndrome.) M. VILLARET and L. TIXIER, *Arch. Gén. de Méd.*, Aug. 1, 1905, p. 1935.

THE authors report two cases observed clinically, one with an incomplete hemiplegia which gradually cleared up, the other unconscious with a flaccid condition of all four extremities and the rapid formation of bed sores, going on to a fatal termination—no autopsy was obtained. In both cases the diagnosis is based on the withdrawal by lumbar puncture of cerebro-spinal fluid uniformly mixed with blood.

COLIN K. RUSSEL.

**SYMPTOMATOLOGY OF CEREBELLAR TUMOURS. A Study of (290) Forty Cases.** T. GRAINGER STEWART and GORDON HOLMES. *Brain*, Vol. xxvii., 1904, p. 522.

THIS work amounts to the most complete and most lucid description of cerebellar symptomatology in man which can be found in the literature. It contains much new and important matter bearing upon the following points :—

The constant relation of the nature of the vertigo to the position of the lesion.

Atonia, diadococinesia, static ataxy.

Compensatory attitudes and over correction.

Cerebellar fits.

Differential diagnosis of subtentorial tumours.

The details of this paper are so essential that they cannot be usefully summarised; the reader must consult the original if he

is to become aware of its importance and to gain knowledge of its contents. It must suffice in this abstract to emphasise those new facts and deductions which the authors have brought to light.

The most constant symptom of both intra-cerebellar and extra-cerebellar tumours is vertigo. In intra-cerebellar tumours the sense of rotation of self is in the same direction as the apparent movement of external objects. In extra-cerebellar tumours the sense of rotation of self is in the opposite direction to that of the apparent displacement of external objects.

This distinction in the sensation of rotation of self and external objects was constant in each class of case, and upon this physical sign alone can the diagnosis be made as to whether a subtentorial tumour is situated within or without the cerebellum. The high importance of this aid in diagnosis *re* surgical interference is obvious.

It is suggested that the "skew" direction is an irritative rather than a paralytic cerebellar phenomenon.

When describing the homolateral atonia of cerebellar disease the authors show that if an attempt be made to execute a movement against resistance in the limbs of the same side as the cerebellar lesion, if the resistance be suddenly removed, the limbs fly rapidly in the direction of the strain, to the anatomical limit of such movement, and there is little or no recoil.

The reeling gait of cerebellar disease is described as being in great measure due to the irregular action (? inco-ordination) of the trunk muscles, since patients who are so unsteady on their feet that they can scarcely walk, may show but slight inco-ordination in carrying out movements of the lower limbs as they lie in bed.

The diadococinesia of Babinski is described as being neither pathognomonic of, nor constantly present in, cerebellar disease, though it is usually well marked.

The remarkable steadiness and absence of the normal vibration when an affected limb is held rigidly extended, to which attention was first drawn by Babinski, has been constantly found by the authors, who consider that this phenomenon emphasises the purely kinetic character of cerebellar ataxy. This sign is only constant in intra-cerebellar tumours. In extra-cerebellar tumours, on the other hand, irregular tremor is often present.

The gait which is peculiar to unilateral cerebellar disease is characterised by two distinct components—

- (1) A tendency to stagger to the side of the lesion.
- (2) A tendency to deviate from the desired line of progression toward the side of the lesion.

The account which the authors give of the compensatory attitudes which the patients regularly assume to correct these



faults, and the tendency to over-correction which may cause an uninitiated observer to fall into error as regards the side of the lesion, are most important expressions of previous knowledge upon this subject.

Attention is especially drawn to the variability of the tendon reflexes in intra-cerebellar disease.

Operations upon the cerebellum invariably caused bilateral loss of the tendon-jerks for some days following the operation. They reappeared quickly and always upon the contralateral side first. In extra-cerebellar tumours it is very rare for the tendon reflexes to be diminished.

An extensor plantar reflex was never present in any uncomplicated case of cerebellar disease.

A very complete description is given of cerebellar fits which occurred repeatedly in two of the cases. The seizures consisted entirely of tonic spasm, the face was unaffected, and respiration was not disturbed. The limbs were rigidly extended and adducted, rotated inwards upon the side of the lesion, rotated outwards upon the opposite side. There was some tendency for the body to turn on to the opposite side.

The authors have not observed cerebellar fits identical with those recorded by Dr Hughlings Jackson, but they are of opinion that the fits they describe were of unilateral origin, whereas those of Dr Jackson were similar phenomena of bilateral origin (the lesions in the later cases were in the vermis).

The differential diagnosis of the three varieties of subtentorial tumours, intra-cerebellar, extra-cerebellar, and intra-pontine, is very clearly described, and symptoms of the three conditions are well contrasted by being placed in a tabular form.

The paper concludes with a comparison of the results obtained by the writers in man with the results of experimental cerebellar lesions in animals by Ferrier and Turner, Risien Russell, Luciani, Thomas, Sergi, and others.

It is suggested that some of the differences in the results obtained in man and the lower animals may depend upon : (1) The greater compensation for cerebellar lesions possible in man, such potentiality of compensation diminishing from the highest to the lower mammals. (2) The erect attitude of man, the change in the place of locomotion and the narrowing of the biped basis. (3) The rate of onset of the lesion ; for with a slowly increasing lesion there is compensation proceeding *pari passu*. JAMES COLLIER.

**FACIAL NEURALGIA AND TUMOUR OF THE GASSERIAN**  
(291) **GANGLION.** (*Névralgie faciale et tumeur du ganglion de Gasser.*) VERGER and GRENIER DE CARDENAC, *Rev. Neurolog.*, July 30, 1905, p. 704.

THERE are a few recorded cases of trigeminal neuralgia due to tumour of the gasserian ganglion. In the author's case a small primary cancer about the size of a bean was found to occupy the left cavum Meckelii.

A diagnosis of tumour is possible from the following considerations:—

1. The neuralgia is constant; there is no relief between paroxysms.
2. The pain is exacerbated by peripheral excitations.
3. All three branches are involved.
4. The corresponding half of the face is markedly hypæsthetic and hypalgæsic.
5. The muscles of mastication are often paralysed.
6. Local injections of cocaine or stovaine have only a slight and transient effect.

It is conceivable that, in a certain number of these cases, the neuralgic phenomena might disappear consequent on the complete destruction of the ganglion or roots.

The authors seem to be under the impression that if a diagnosis of tumour be made, surgical interference is unwarranted.

S. A. K. WILSON.

**A CASE OF CHOREA FATAL FROM ACUTE STAPHYLOCOCCAL**  
(292) **MENINGITIS.** (*Un cas de chorée mortelle par méningite aiguë à staphylocoque.*) E. LEONÉ and GAUDEAU, *Bull. de la Soc. de Pédiatre de Paris*, May 1905, p. 149.

THE authors describe a case of chorea in a boy of 10, which at first appeared to be an ordinary though severe case of the disease. The child did not improve under treatment, and 17 days after the onset the symptoms became aggravated and the temperature rose. Three days later the movements diminished, but the child became torpid and did not reply to questions. Two days later slight rigidity of the neck was noticed, and lumbar puncture confirmed the suspicion of meningitis. The symptoms persisted, and after other three days the child died.

The urine during the illness contained no sugar or albumen; the urea, chlorides, and phosphates were not altered; the uric acid was increased.

The fluid removed by lumbar puncture was normal at the date of admission, but at the time when rigidity of the neck was noticed it was albuminous and contained numerous leucocytes, chiefly polynuclear. A fine culture of staphylococcus aureus was obtained from the fluid.

The authors think that infection of the meninges is probably less exceptional in fatal cases of chorea than has been supposed, and furnish arguments in support of this opinion.

W. B. DRUMMOND.

**THE PHYSIOPATHOLOGY AND THEORY OF TIC.** (Considerazioni su la fisiopatologia e su la teoria del tic.) SICCARDI, *Annuario del Manicomio Provinc. di Ancona*, Vol. ii., 1904.

CONSIDERABLE misconception exists as to the exact meaning and scope of the word tic. Within recent years there has been, more especially on the Continent, a renewal of interest in the subject, which has led to widespread discussion and painstaking analysis of the phenomena. It has been made abundantly evident that many essentially differing pathological conditions have been hopelessly confused. The word has been too loosely employed to include various forms of spasm, twitching, and reflex motor reaction, as well as all sorts of tricks and mannerisms.

Innumerable definitions have been given of the word. To say that it is an "imperative movement," a "parasitic function," is to use a mere paraphrase. It is more than a simple spasm, as Brissaud has been at pains to indicate. True it may originate in a spasm, but the latter is essentially a reflex movement due to irritation at some point in a reflex spinal or bulbo-spinal arc, and it is only when the cortex intervenes that the possibility of its becoming a tic arises.

In every instance a tic has been originally a purposive act evoked by some definite cause, and directed to some definite end. Its frequent repetition, however, when all excitation has ceased, is the first step in its degeneration; and when this repetition has become meaningless and inopportune, yet irresistible and urgent, the tic is full-blown.

Not all who would may tic, Charcot was in the habit of declaring, and this leads to another feature in the disease: it occurs only in the mentally unstable. It is owing more especially to the work of Meige and Feindel that this fact has been properly emphasised. The subject of a tic betrays mental infantilism in greater or less degree. The diminution of his power of inhibition is sufficient evidence of the truth of this statement.

It is instructive to differentiate between the tics and the move-

ments of distraction called by Seglas "stereotyped" acts. The innumerable tricks we reveal during any mental or physical exertion are stereotyped acts, not tics, however inapposite, meaningless, and involuntary they be. They occur only when the attention is diverted; whenever we turn our mind to them they can be checked at once. Now it is precisely in the absence of any physical or mental exercise that the tic runs riot; any voluntary inhibition of it is purely momentary; and many instances are on record where occupation of the mind in other ways sufficed to stop the movements altogether.

S. A. K. WILSON.

**WRITER'S CRAMP AND WRYNECK OF MENTAL ORIGIN.**

(294) (*Crampe des écrivains et torticollis d'origine mentale.*) G. BONNUS, *Nouv. Icon. de la Salpêtrière*, May-June 1905, p. 285.

DR BONNUS describes a case of writer's cramp and subsequent torticollis in a woman of fifty, very emotional, but with none of the stigmata of hysteria. The cramp first appeared after an attack of influenza, and lasted intermittently for ten years. Several temporary faith-cures were effected (by prayer, etc.), but it ultimately was constantly present, the patient becoming possessed with a fixed idea that her hand was weak and that cure was impossible.

The torticollis followed on a stiffness of the neck, in which relief was obtained by turning the head to one side, which movement soon became involuntary.

Both conditions were successfully relieved, mainly by psychotherapy, combating the fixed ideas of weakness and incurability.

J. H. HARVEY PIRIE.

**PSEUDO GENERAL PARALYSIS OF DIABETIC ORIGIN.** (La

(295) *pseudo-paralysie générale diabétique.*) INGEGNIEROS, *Rev. Neurolog.*, July 30, 1905, p. 709.

IN this paper the contention is made that general paralysis is not so much a disease *per se* as a symptom complex common to various forms of meningo-encephalopathy. Its symptomatology depends on the site of the lesion, not on its nature. The syndrome may arise accidentally in the course of some intoxication, or it may be produced by a special localisation of syphilis, saturnism, alcoholism, etc.—the pseudo general paralyzes. Or it may be of the progressive incurable type, leading to dementia paralytica.

No one of the symptoms in this syndrome—mental, motor, pupillary, trophic,—is in itself pathognomonic of the condition. Each and all of them, further, may arise in the course of diabetes.

The case described presented the typical clinical picture of general paralysis, and was at the same time a diabetic. He was constantly under observation during a period of three years, in which time ample evidence was obtained of the remission of the general paralytic symptoms by suppression of the diabetes.

A diabetic auto-intoxication affecting simultaneously the various nerve centres, lesions of which produce general paralysis, ought to reveal itself by this syndrome, so that diabetic pseudo general paralysis becomes a real clinical fact. S. A. K. WILSON.

**EARLY AND PERMANENT CONTRACTURES IN A CASE OF  
(296) HEMIPLEGIA IN THE ADULT. (Contractures précoces et  
permanentes dans un cas d'hémiplégie de l'adulte.) A.  
GAUSSEL, *Nouv. Icon. de la Salpêtr.*, May-June 1905, p. 241.**

CONTRACTURES in hemiplegia are well recognised; their pathogenesis is still open to discussion. They may come on early or late, at the time of the occurrence of the lesion, or days or weeks later. According to classic authorities the two forms are clearly differentiated. The first class usually accompanies the participation of the meninges in the lesion or the flooding of the ventricles. The late contractures follow descending degeneration of the pyramidal tract. Dejerine has pointed out that the early contractures are usually transient, but in certain cases they persist and become permanent. Besides the hemiplegia of the adult, one ought to mention the contractures and bizarre attitudes observed in infantile cerebral hemiplegias, in which the extremities particularly are often the seat of athetoid movements. As a rule the contractures seen in the adult form have a quite distinctive character from the permanent contractures of the infantile cerebral hemiplegic type. The author reports a case of R. hemiplegia with aphasia, in which the contractures of the wrist and hand simulate an infantile paralysis, while in reality it occurred in a woman aged 62, and was complicated by the occurrence of early and permanent contractures. It was considered due to a cortico-meningeal hæmorrhage, involving and destroying the L. perirolandic region in the posterior part of the third frontal convolution. The article is illustrated by two photographic reproductions.

COLIN K. RUSSEL.

**A CASE OF ARTHRITIC MUSCULAR ATROPHY.** (*Un cas (297) d'atrophie abarticulaire.*) Dr A. DEROUBAIX, *Journal de Neurologie*, July 5, 1905, p. 241.

THE author reports a case of a man who for sixty years had ankylosis of the knee-joint, following a probable tuberculous inflammation in youth.

Histological examination shows the *muscle* fibres markedly atrophied, but not degenerated, since they retain the transverse and longitudinal striation. One is struck by the great increase of nuclei both under the sarcolemma and also in the muscle substance. The vessels show no endarteritis, and in general their lumen is filled with red blood cells. The external popliteal nerve is normal.

In the spinal ganglia one is struck with the marked dilatation of the vessels and with the great variation of colouring and volume of the cells. One sees every grade between the large, almost colourless cell and the small, deeply-stained cell. In some of these small cells the nucleus is ectopic. It is difficult to say whether this is merely due to functional activity or to a pathological peripheral irritation. Most of the cells are pigmented, in some only at one edge, in others concentrically. The nucleolus stains clearly and is surrounded by a clear zone.

In the sacral cord the anterior horn cells are normal, but in the posterior horn and in the intermediary zone on the right side the cells show chromatolysis and marked ectopia nuclei. The same change is noted in the column of Clarke in the dorsal cord.

The recent works of Langley, Onuf and Collins, Cassirer, Huet, Bervoets and others, show that the sympathetic system takes origin from cells about the central canal, between the anterior and posterior horns, and that part innervating the lower extremity is derived from the second and third sacral segments. Its fibres accompanying the peripheral nerves subserve the regulation of the vascular supply and the maintenance of the trophic condition of the part.

Nerves subserving the sensibility of the joint play an important part in the function of equilibration and co-ordination, and are in close relation with the cerebellum. On the other hand, it is rational to suppose that there is a reflex arc between these articular fibres and those charged with the maintenance of the trophic condition of the part, and consequently an irritative articular condition may, through this reflex sympathetic arc, set up atrophic conditions of the part. Giacomo Pighini claims to have shown this experimentally in rabbits.

The author considers that the lesion must be explained on this

neuro-irritative theory. He suggests that myopathies, the arthritic condition of tabes and syringomyelia, and other obscure chronic arthritic changes may be explained on the same theory.

COLIN K. RUSSEL.

**FATAL HYPOTONIC MYASTHENIA.** (*Myasthenie hypotonique* (298) *mortelle*.) E. DUPRE and P. PAGNIEZ, *Nouv. Icon. de la Sal-pêtr.*, May-June 1905, p. 247.

THE authors report a case of myasthenia, with sudden cardiac failure and death. At the autopsy the organs were found to be healthy, the thymus gland was present and weighed 8 grammes, but presented nothing pathological microscopically. Examination of a portion of the thyrohyoidian muscle showed the fibres well preserved in size and form, and well stained. There was no sign of degeneration and no indication of inflammatory reaction, only an insignificant increase in the number of nuclei. The pneumogastric and phrenic nerves, as well as the rest of the nervous system, were negative. The case differed from the usual type in two points, first, in the contrast between the general spinal character of its clinical course and the unexpected bulbar nature of the fatal termination.

Secondly, in the presence of a marked hypotonia of the muscles, comparable with that of tabes, with no lesion of the nervous system to account for it. The article is illustrated by several photographs.

COLIN K. RUSSEL.

**A CASE OF VESTIBULAR ATAXIA.** (*Un cas d'ataxie vestibulaire* (299) *laire*.) RAYMOND and EGGER, *Rev. Neurolog.*, June 30, 1905, p. 610.

THE patient was a woman, aged 42, suffering from a bulbar lesion involving the fifth, sixth, seventh, eighth, and ninth cranial nerves on the right side. When she stood with her feet together, oscillations began in the upper part of her body, increasing in intensity with the closure of her eyes till she fell, which she did always to the right. Her gait was highly irregular; sometimes she would take several quick steps in advance; sometimes she would hesitate or even go back a little; her paces were unequal in length and quite arhythmical. With eyes shut it became obviously worse; a forced lateropulsion seemed to make her fall always to the right.

An interesting experiment was made by placing the patient on a chair which was fixed to a board revolving round a central axis. When the apparatus was revolved to the left, the patient being seated with her face towards the periphery, she was at once conscious of this rotation to the left; on checking the movement she did not experience the illusory sensation of rotation in the opposite direction which is at once detected by the normal individual. On turning the chair to the right she did not know that she had been displaced at all until the rotation was stopped, when she immediately remarked that now she seemed to be going to the left.

These observations indicate destruction of the vestibular mechanism on the right side, and in the authors' opinion it is unnecessary to postulate further lesions to account for the clinical symptoms, since similar results have been obtained by experimentation on the semicircular canals of animals.

S. A. K. WILSON.

**L'ASCHEMATIE.** PIERRE BONNIER, *Rev. Neurolog.*, June 30, 1905, (300) p. 605.

By the word *aschematie* the author signifies the affection whereby certain parts of the body cease to be represented in the general idea which we have of the body as a whole. It is anæsthesia limited to position in space, to distribution, form, position, attitude; it is, in short, topographical anæsthesia.

The term is preferable to the kinæsthesiopathy of Deny and Camus.

A few clinical examples will convey best the author's meaning. One patient had attacks in which there was momentary suspension of all conscious localisation in the sensory field. "During these moments I continue to walk along, and continue to feel, but nothing seems to be anywhere, and I myself don't seem to be anywhere." Another patient with Meniere's disease used during the attacks of vertigo to lose all sense of his own personality. A third had complete transient loss of the faculty of orientation. The result is that everything for the time being loses its reality. Muscular activity has nothing to do with the condition; nor is there any interruption of ordinary sensation; what is wanting is the reference to a position in space of an object, the sensation of which persists.

A portion of the communication is taken up with the question of priority.

S. A. K. WILSON.



**ACUTE AND CHRONIC CIRCUMSCRIBED ŒDEMAS UNDER THE**  
**(301) INFLUENCE OF THE NERVOUS SYSTEM.** (*Les œdèmes*  
*circonscrits et chroniques sous la dépendance du système*  
*nerveux, etc.*) VALOBRA, *Nouv. Incon. de la Salpêtr.*, May-June  
 1905, p. 255.

*Etiology.*—Quinke's disease occurs most frequently in neuro-pathic individuals, often with some hereditary taint, as numerous cases have been recorded in which there was a history of the condition in several generations. It is seen also in the course of such intoxications as ethylism, carbon monoxide poisoning, thyroidism, etc. Some would distinguish Quinke's œdema from urticaria, affirming that the former never supervenes during intestinal troubles, but a case is recorded in this paper which is direct evidence to the contrary. Besides, Quinke's œdema is by no means an uncommon phenomenon in infective diseases.

*Symptomatology.*—The classic description is that on the skin appears a circumscribed œdema of varying size, neither irritable nor painful, which disappears after a few minutes or hours. Numerous exceptions have been observed, however. The colour is usually pale, but it may be very red. The distribution is sometimes irregular, sometimes segmental. There may be slight objective alteration of sensibility over the area. The local temperature is below that of the surrounding skin.

*Diagnosis.*—At the present time, thanks to the influence of German teaching, urticaria is reckoned a disease of the skin, whereas circumscribed œdema is a disease of the nervous system. Nothing could be more preposterous. There is not a single one of the morphological features of urticaria which is wanting in the cutaneous phenomena of Quinke's œdema. Innumerable cases of the association of the two have been recorded. The remarkable fact that each may be accompanied by hæmoglobinuria is not without significance. In exophthalmic goitre, acroparæsthesia, peliosis rheumatica, Reynaud's disease, syringomyelia, paludism, etc., the combination of the two has been noted.

It may be said that urticaria, Quinke's œdema, and the troph-œdema of Meige occur under identical circumstances and for identical reasons in individuals constitutionally predisposed; they may simultaneously manifest themselves in a patient; and transitional forms are so constantly met with that it is impossible to find a single essential point of difference.

That all three conditions are under the influence of the nervous system cannot be doubted. Clinical and experimental evidence is abundantly forthcoming; it is impossible in an abstract to multiply instances.

*Pathogeny.*—The anatomical and physio-pathological basis of urticaria and of circumscribed œdema is the excito-secretion of lymph, associated or not with vaso-dilation. The alteration is transient, as the lymph is quickly reabsorbed, but in trophœdema and in elephantiasis the change in the lymphatic circulation consequent on the stasis of the lymph secretion results in hyperplasia of the connective tissue, *i.e.* the deformation is permanent.

The author considers that there is evidence for the existence of medullary lymph secretion centres metamerically arranged.

S. A. K. WILSON.

**QUINQUAND'S SIGN.** HOFFMAN and MARX, *Berlin. klin. Woch.*, (302) May 1905.

HOFFMAN and Marx contribute an article on Quinquand's sign and its significance as a diagnostic indication of alcoholism. They review the work of previous investigators, considering that the best description of the phenomenon is given by Fürbringer (*Deutsch. med. Woch.*, 1904, No. 27). "If the outstretched finger of the person under observation be placed upon the palm of the investigator's hand, after two or three seconds slight movements occur as if the finger-bones were rapidly striking against each other and against the observer's hand." On auscultation Fürbringer found that a distinct crepitation was audible, varying considerably in intensity.

In a series of investigations on 1018 prisoners, the authors came to the following conclusions:—

1. Absence of the sign or its presence in moderate intensity does not warrant the conclusion that the subject is an abstainer. At the highest computation the likelihood can only be placed in the proportion of 3 : 2.

2. Its presence in intense degrees points with a probability of 3 : 1 to confirmed drunkards, and in the proportion of 2 : 1 to moderately heavy drinkers.

These results differ materially from those of Fürbringer and Levienik, but the authors consider it probable that the difference arises chiefly from the fact that not only were the patients examined by them more numerous, but the proportion of heavy drinkers was noticeably higher. They were unable to trace any definite connection between the sign and other neuroses, some hysterical and neurotic patients showing no trace of it, whilst in others it was evident.

They confirm Fürbringer's opinion that the phenomenon is of less frequency and intensity in women, and that the curve of frequency varies with the age of the patient, rising to its height at maturity and then decreasing with advancing years.

Throughout the examinations the sign followed a regular course, gradually increasing to a height, then diminishing to complete cessation, reappearing after a short interval of rest.

The effect of the enforced abstinence of prison life was apparently inconclusive—in some cases the sign diminished after a few months, in others it showed no change.

In the opinion of the authors the sign is of greater value in the diagnosis of alcoholism than either tremor of the hands or tenderness of the calf muscles, and must always be a weighty factor in the list of symptoms.

There seems as yet to be no evidence of any definite connection between tremor and the presence of Quinquand's sign—many cases with very marked tremor showing no trace of it, and, on the other hand, many cases with intense degrees of "Quinquand" exhibiting no tremor. The authors consider that the muscular action in either case may be quite different in nature, and they advance the hypothesis that Quinquand's sign may result from the contraction of individual muscle-bundles, whilst tremor indicates the action of the whole muscle, the muscular irritability induced by alcoholism being in each case a predisposing cause.

The authors found that it was possible to produce Quinquand's sign by experiments on persons in whom it was not normally present, especially if irritability of the muscles of the arm were induced by the application of the faradic current, and conclude by pleading the necessity of further observation of this interesting phenomenon.

E. M. ERSKINE.

(303) **QUINQUAND'S SIGN.** HERZ, *Munch. med. Woch.*, May 1905.

HERZ has a short article on the Quinquand's sign. The greater part of the paper deals with the question of the site of production of the auscultatory sound. In his investigations, Herz adopted a modification of the ordinary method of auscultation, being of the opinion that the latter is not very well adapted to the special circumstances. He employed for the purpose a piece of elastic rubber-tubing—about 2 cm. wide—closing it at one end with a cork and connecting the other end with an ear-piece. The corked end was laid upon the table, the ear-piece inserted in the ear of the investigator, and the person under observation was instructed to rest his finger on the rubber tube. By this method Herz was able to detect the slightest "crepitation."

He does not agree with Fürbringer that the sound is produced in the finger-joints, but believes that it is occasioned by very slight separations of the digital-flexor tendons from their sheaths—the tendons being at the time in a state of tension. In a series of experiments he found that the quality of the sound varied con-

siderably with the position of the finger, becoming fainter in proportion to the increase of flexion, and increasing in intensity as the finger was again extended. Passive or voluntary movement could also in certain circumstances induce an augmentation of the sound. Herz suggests the term "Tendon vibration" (*Sehnenschwirren*) as descriptive of the condition. He also investigated the large joints, but failed to obtain any result, except in the single instance of the knee, where he was able to elicit, experimentally, a similar phenomenon in the tendon of the quadriceps extensor.

He summarises his conclusions as follows:—

(1) "Tendon-vibration" (*Sehnenschwirren*) can be evoked in every person by very slight, voluntary, spontaneous, or passive movement of the digital-flexor tendons in their sheaths, or of the patella on its articular surface.

(2) Quinquand's Sign does not originate in the joints, but is a spontaneous vibration of these digital flexors in their sheaths, evoked by involuntary muscular contraction.

(3) The general muscular restlessness characteristic of alcoholism, may account for the increased tendency to the phenomenon in this condition.

E. M. ERSKINE.

## PSYCHIATRY.

**BRAIN WEIGHT AND MENTAL DISEASE.** (*Hirngewicht und (304) Geisteskrankheit.*) R. MITTENZWEIG (of Herzberge), *Allg. Ztschr. f. Psych.*, Bd. 62, Heft 1, 2, with 18 tables.

MITTENZWEIG gives the results of a research into the relation between mental disease and brain weight. The material included 1123 brains of patients whom he divides into 4 groups—general paralysis (293 M., 174 F.), senile dementia (124 M., 208 F.), organic psychoses (87 M., 52 F.), functional psychoses (81 M., 104 F.). He reaches the following conclusions:—

A brain weight of under 1000 g. in a male, whether over or under 60, renders probable the presence during life of mental disease—probably dementia paralytica, dementia senilis, or (if the individual be over 60) an organic psychosis.

If the brain of an insane male patient be under 1100 g. (in a patient over 60), or under 1150 g. (in a patient under 60), it is unlikely that the case was one of a functional psychosis.

A brain weight of under 1000 g. in a female under 60 makes mental disease during life fairly certain—either dementia paralytica or an organic psychosis.

A brain weight of under 950 g. in a female over 60 makes mental disease during life certain—either dementia senilis or an organic psychosis.

C. MACFIE CAMPBELL.

**AMENTIA AND DEMENTIA: A CLINICO-PATHOLOGICAL**  
(305) **STUDY.** JOSEPH SHAW BOLTON, *Journ. Ment. Sci.*, April and  
July 1905.

THESE papers contain further instalments of the writer's researches on the general pathology of mental disease, and are the first two of a series. The series of papers is divided into three parts, of which the first is concerned with clinico-pathological data bearing on the morbid anatomy of dementia, and the second and third with the clinical aspects of amentia and dementia respectively. The papers at present under consideration deal with Part I. and a portion of Part II.

PART I. This paper is devoted to the general verification and amplification of the results obtained during the writer's study of the morbid anatomy of dementia. To the 200 cases previously published a further 233 have been added, and these are classified into the five groups previously made use of, namely, (1) cases without dementia; (2) cases with appreciable dementia; (3) cases of insanity with moderate dementia; (4) cases of dementia which still show symptoms of insanity; and (5) cases of gross dementia. These groups agree closely with the following five classes, which are based on morbid appearances: (1) cases without morbid changes and where the pia arachnoid strips naturally; (2) cases with slight morbid changes and where the pia arachnoid strips rather more readily than natural; (3) cases with moderate morbid changes, with sub-dural excess to the level of the tentorium and where the pia arachnoid strips readily; (4) cases with marked morbid changes and where the pia arachnoid strips very readily; and (5) cases with very marked morbid changes and where the pia arachnoid strips like a glove from the hemispheres. The morbid appearances found in these groups are analysed and summarised, and separate sections are devoted to two important conditions, which, in certain respects, modify the morbid anatomy of dementia, namely, degeneration of the cerebral vessels and systemic tuberculosis.

The result of the writer's clinico-pathological and histological studies is a classification of mental diseases based on the thesis that these diseases consist in essence of a large group of cases with varying degrees and types of cerebral degeneracy, and of another larger group with varying degrees and types of cerebral dissolution. To the former group he applies the term *amentia*, which thus connotes in the widest sense *the mental condition of patients suffering from deficient neuronie development*; and to the latter the term *dementia*, which similarly connotes *the mental condition of patients who suffer from a permanent psychic disability due to*

*neuronic degeneration following insufficient durability.* The second and third parts of the research deal respectively with these two groups of cases from the clinical standpoint, and contain a descriptive classification of a series of 728 chronic and recurrent lunatics which is elaborated from personal notes made on the cases by the writer.

**PART II. AMENTIA.** Of the 728 cases made use of during the research, 283 fall under the category of amentia, and 445 under that of dementia. The cases of amentia are subdivided into two groups—*low-grade aments*, or idiots and imbeciles, of which there are 94, and *high-grade aments*, of which there are 189.

*Low-grade amentia.* The 94 cases in this group are briefly analysed and classified, and the description is illustrated by average examples of the different types.

*High-grade amentia.* The 189 cases in this group are examples of mental disease which possess a milder degree of cerebral under-development and mental deficiency than the imbecile, and which form the connecting link between the mildest type of imbecile on the one hand and the ordinary "sane" individual of average intelligence on the other. This group of cases closes a recognised but hitherto unfilled gap between morbid and normal psychology, and makes it possible to reduce the subject of mental alienation into a coherent system, which, on the one side of the normal, includes all types possessing deficient neuronic development under the term "amentia," and on the other side all cases suffering from deficient neuronic durability with resulting degeneration under the term "dementia."

This group contains many cases which are usually classified as "chronic mania" or "chronic melancholia," but which exhibit developmental deficiencies, which show themselves under the form of general mental or moral instability or perversion on the one hand, or under that of curious and abnormal mental function on the other. It also includes recurrent cases of mental disease, whether these are still subject to periodic relapses and are temporarily under treatment, or are permanently certified. Further classes in the group contain cases of hysteria and of true epileptic insanity. Finally, the writer has inserted cases of insanity with systematised delusions (including paranoia), and he believes and hopes to demonstrate that these also are developmental in origin and should be described amongst the highest types of amentia.

These cases possess a common physical feature, namely, a more or less marked under-development of the cerebrum associated with the absence of intra-cranial morbid appearances, and a common psychic feature, namely, the entire, or almost entire, absence of dementia, unless this occurs as the result of senile or

presenile involution of the cortical neurones, or in a small proportion of cases as the result of neuronie degeneration following excessive primary and direct toxæmia of the cerebrum.

Of these cases, the present contribution deals with the first class only, namely, "excited and 'moral' cases," of which there are 22 males and 64 females. Although the cases in this class belong to one defined group, it has been found desirable to further subdivide this into four sub-classes, namely :—

	M.	F.	T.
(a) "Moral" cases . . . . .	5	9	14
(b) Simple emotional chronic mania . . . . .	4	32	36
(c) Chronic mania with incoherence and delusions . . . . .	6	13	19
(d) "Cranks" and asylum curiosities . . . . .	7	10	17
	<hr/>	<hr/>	<hr/>
Total . . . . .	22	64	86

The cases in these sub-classes show, respectively, the following prominent characteristics :—

- (a) *Alteration of moral sense*, with a tendency to do desperate things, *e.g.* to commit suicide or even homicide, to perform acts of self-injury or self-mutilation, to strike, smash, or destroy, to intensely irritate those around them, to be sexually inclined in a normal or abnormal manner, etc.
- (b) *Alteration of emotional and intellectual control*, *e.g.* exuberance, instability, vanity, garrulity, childishness, and often violence, treachery, and destructiveness. The younger and adult types usually display a more or less marked loss of control over the emotions and instincts. The older types differ from these in the fact that the loss of control affects chiefly the intellectual functions. Their association of ideas is normal, except for its extreme rapidity and complexity. They talk continuously whenever a listener can be found, and they are frequently inconsequent and show a marked tendency to parenthesis during their descriptions.
- (c) *Rapid and uncontrolled association of ideas*, with delusions of grandeur, which may or may not co-exist with or follow delusions of persecution. These cases form a half-way house between sub-classes (b) and (d), and shade gradually into each of these. They differ from the former in being, on the whole, less troublesome, and in showing an apparently complete incoherence in their association of ideas, and from the latter in the fact that their ideation is simply rapid and uncontrolled, rather than grotesque or symbolical and resulting in erratic and eccentric conduct.

- (d) *Stereotyped, symbolical, or grotesque association of ideas*, which leads to weird actions and eccentric general behaviour. These cases are extremely conceited, vain, and grandiose. They are of many types, and may be simply asylum "show-birds," or may possess considerable artistic or intellectual talent. As a class, these cases only differ from certain "sane" individuals in the absurd and grotesque extremes to which they carry their ideas and their resulting behaviour and actions; and their stereotypism, which often suggests dementia, also only differs in degree from the stereotypism and prejudice which are often seen in the "cranks" of the outside world.

The descriptions of these several sub-classes are illustrated with average examples of the different types.

AUTHOR'S ABSTRACT.

**DEMENTIA PRÆCOX OR BRAIN TUMOUR?** (*Dementia præcox* (306) oder Gehirntumor?) O. KAISER (of Neufriedenheim), *Allg. Ztschr. f. Psych.*, Bd. 62, Heft 1, 2.

PATIENT was a young woman of bad heredity, but healthy, and of apparently good mental constitution until the age of 22 (1892), when mental symptoms came on abruptly. She suddenly became almost mute, refused to dress herself, was seclusive, without initiative, tearful and anxious; after a short period, she passed into a condition of stupor which lasted for several months. Mutism, negativism, stereotyped utterances were prominent symptoms. Patient soiled herself, had on one occasion to be catheterised; she retained saliva in her mouth. The stupor gave place to a condition of excitement, which persisted over a year before it subsided somewhat, and began to alternate with periods of quietness and comparative clearness, patient gradually deteriorating. Occasionally patient was erotic; Ganser's symptom (random answers) was observed: she called herself high-sounding names, said that she was six years old, that her mother was 26 (her own age); she called a mole her new-born child.

From 1897 deterioration rapidly progressed, and patient finally presented the appearance of profound dementia. She continually soiled herself, apathy alternated with excitement, she resisted attentions, her only utterances were inarticulate shouts. The course and clinical picture of the case justified the diagnosis of dementia præcox.

In 1902 the situation suddenly altered: patient developed attacks of Jacksonian epilepsy, beginning in the face and arm of



the right side, occasionally affecting the right leg and extending to the left side: in 1903 the right arm became paralysed. There was no choked disc. Patient died September 1903.

The autopsy disclosed a diffuse glioma spreading through the whole of the right hemisphere with hæmorrhages and foci of softening; also pachymeningitis and leptomeningitis over the whole of the convexity of the brain.

The fact that a tumour of this extent had not produced marked pressure symptoms could only be explained by a very slow development, and the gradual progression of the clinical symptoms justified the view that the symptom-complex of dementia præcox had been produced by the tumour itself.

C. MACFIE CAMPBELL.

#### **ENDOGENOUS SYMPTOM-COMPLEXES IN EXOGENOUS FORMS**

(307) **OF DISEASE.** (*Endogene Symptomenkomplexe bei exogenen Krankheitsformen.*) FAUSER (of Stuttgart), *Allg. Ztschr. f. Psych.*, Bd. 62, Heft 1, 2.

FAUSER reports the case of an actor aged 35, who suddenly developed a typical picture of the manic phase of manic-depressive insanity—motor unrest, elated mood, great loquacity with marked flight of ideas, sound-associations, distractibility—with absolutely no symptoms pointing to general paralysis. After a year patient had markedly improved, although he still showed some restlessness, loquacity, and distractibility, and was discharged to the care of his father, a physician; at the time of his discharge there was no speech defect nor pupillary symptoms. Four months later patient had an epileptiform attack, his pupils were irregular and reacted sluggishly, his speech was involved, and the further course was that of a typical case of dementia paralytica.

The author calls attention to the frequent occurrence in general paralysis and other psychoses not only of symptoms of manic-depressive insanity, but of symptoms of neurasthenic and hypochondriacal natures. Hysterical, neurasthenic, and manic depressive complexes are distortions of normal mental life on a degenerative basis: they are closely akin to tendencies present in all, but restrained through education and habit. The occurrence of the manic complex in the case of a general paralytic may be looked upon as due to the elimination of these acquired checks by the morbid process of general paralysis, and is therefore a defect symptom similar to that observed in the other products of education, habit, and intellectual effort.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO OUR KNOWLEDGE OF COLOUR PER-  
(308) CEPTION.** (Zur Kenntnis des Farbensehens.) G. LOMER (of  
Neustadt-Holstein), *Allg. Ztschr. f. Psych.*, Bd. 62, Heft 1, 2.

THE subjective perception of objects as coloured may occur after the use of certain drugs, or of electricity, and after head trauma; also in fevers, certain neuroses and psychoses.

After a review of cases hitherto recorded, the author gives the observation of a girl with bad heredity, who from childhood had shown psychasthenic traits, suffering from headaches and conditions of anxiety. At the age of 17 she had a hallucinatory attack, with fits of anxiety, which seem to have come on episodically; for the next four years she seems to have had a series of such attacks, and in the intervals to have been unable to adjust herself to her environment. She showed no signs of deterioration at the end of this time. At the age of 21, after a clear interval with good insight, patient suddenly had an attack of excitement, which soon passed off, when she was isolated. During the attack patient complained of seeing everything green, and after the attack this symptom persisted. For two weeks she continued to have attacks daily, and for two weeks longer she continued to see everything green; the condition then abruptly ceased.

The author inclines to the diagnosis of dementia præcox, although much seems to point to the hysterical nature of the psychosis.

C. MACFIE CAMPBELL.

**ON A FORM OF ABERRANT HYPOCHONDRIA DUE TO THE  
(309) LOSS OF CONSCIOUSNESS OF THE BODY.** (Sur une forme  
d'hypochondrie aberrante due à la perte de la conscience du  
corps.) DENY and CAMUS (of Paris), *Rev. Neur.*, May 15, 1905.

PATIENT was a woman aged 47, who for some years had suffered from attacks of mental trouble, the onset of which was connected with domestic worries. The last attack, which had already lasted two years, was similar to previous attacks. It began with a period of confusion and perplexity, during which patient left her house, wandered about, undressed on the street, attempted suicide. The confusion passed off in several days, leaving patient in a state of marked anxiety, which was due to her altered consciousness of her person and of the outside world. She said: "I do not feel myself as I used to. I no longer feel my limbs, my head, my hair; I must touch them to know how I am. . . . I feel lost. I try to think, and I cannot represent myself. It is terrible, this insensibility; it makes me feel like a blank."

Patient was not conscious of the position of her limbs in bed: to become so she had to touch them or rub one against the other. She could not remain decently dressed, as she felt continually the necessity of feeling the bare skin to assure herself of her own bodily existence. The world and the people in it appeared phantoms, awaking no warm emotion. Her memories shared the same ghostly fate.

Objectively there was no impairment of general sensibility, nor of the special senses. The reflexes were normal; there was no conjunctival nor pharyngeal anæsthesia, no retraction of the field of vision. Mentally, patient's judgment was unimpaired; she had no delusions, but her confinement in an asylum was necessary, owing to her obsession, which made her unable to remain decently clad, and owing to the anxiety which led her several times to attempt suicide.

In discussing the case the authors reject Janet's theory of the mental mechanism involved. Janet holds that the feeling of reality depends on a certain level of psychic tension, and when this is reduced the symptoms under discussion may appear. They favour the explanation offered by the school of Wernicke. In each sensation, whether of the special senses or of general sensibility, there are two elements, a specific and an organic. The former is elaborated into perceptions and our grasp of the external world; the latter gives the warm feeling of reality and personal participation, and it is upon the organic element of the sensations, coming not only from the special sense-organs but from every part of the body, including the viscera, that our consciousness of our own personality is based. In other words, the "*Somatopsyche*" or "*cénesthésie*" is composed of these elements.

The dissociation of the two elements involved in sensation may lead to a splitting of the personality, as in the case of Juliusburger (*vid.* abstract in *Rev. of Neur. and Psych.*, July 1905). The absence of the organic element may lead to a negation of one's personality, with no impairment of perception, as in the present case. The term hypochondria has been limited to cases where there is a disturbance of the organic element in the sensations connected with our vegetative life. In the case under discussion the disturbance is of the sensations connected with our relation to the external world.

In order to retain the term hypochondria in its traditional application, the authors call their case an "aberrant hypochondria," the essence in both being the same, "a function of the *Somatopsyche*" (or *acénesthésiopathie*). Both belong to the group of "*délires cénesthésiques*."

C. MACFIE CAMPBELL.

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# Review of Neurology and Psychiatry

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## Original Articles

### **AN OUTLINE OF ACUTE BERI-BERI AND ITS RESIDUAL PARALYSIS.**

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(Late Director Department for Medical Research, Federated Malay States);  
Beri-beri Research Commissioner, London School Tropical Medicine;  
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IN beri-beri we have a disease, the developed symptoms of which are traceable to the direct action of probably a bacterial toxin on the peripheral terminations of sensori-motor and autonomic neurons. For this reason its pathology is of special interest to the neurologist.

I have elsewhere given in great detail the observations upon which this conclusion is based (1, 2, 3, 4, 5), but as they are not readily available I propose here to outline the clinical and anatomical features of beri-beri, and then briefly discuss its pathology.

Beri-beri is undoubtedly an acute infectious disease with a short incubation period. The invasion in what I have termed the Subacute form is insidious. Cases present with a slight pre-tibial œdema, and sensori-motor disturbance, generally confined to the legs, without being able to tell whether or not such symptoms were preceded by others.

What I have classified as the Acute Pernicious and Acute forms are ushered in with more or less obvious symptoms of gastro-intestinal irritation. These symptoms generally drive a case to the physician at once, if he is convenient. The course of the disease may therefore be watched from its beginning. Malaise

is complained of, and there may be a disinclination to work. Hot or cold flushes may be mentioned. The patient is distressed by, or may casually complain of, cold, or heavy, or burning, or hot sensations, which he refers to the gastro-duodenal region. He may indicate quite definitely that these sensations are felt beneath the right upper epigastric region. Pressure over the pyloric end of the stomach and duodenum intensifies the sensations and is resented by the patient. The throat reddens and may even grow livid. During the presence of the gastro-duodenal sensations there may be a slight pyrexia. On the other hand there may be no rise of temperature at this or any other stage of the disease. In some cases there is a transient catarrhal diarrhoea, in others transient constipation.

It is but natural to suppose that such symptoms are due to an acute indigestion. This mistake is often made in the tropics, and patients are dismissed with remedies to meet such a condition. If kept under observation, however, one's mind is quickly disabused, for in a few hours or days the gastro-duodenal symptoms are followed by loss of the knee-jerks, and by a diminution of voluntary power which, beginning in the peroneal and anterior tibial muscles, may rapidly spread to the rest of the legs, to the thighs, hands, arms, shoulder girdle, trunk, and face. The diaphragm, pharynx, larynx, and even external and internal ocular muscles may not escape. Various degrees of subjective and objective sensory disturbance set in. First in the dorsa of the feet, whence they march with the motor paresis, or they may not extend beyond the knees. A pretibial oedema arrives with the sensori-motor symptoms and may develop into a general anasarca. Evidences of cardiac paresis are amongst the first, if not the first, to appear. In a few days we have to deal with a developed case of acute beri-beri.

The disease then follows one of three courses.

*First.*—If the onset has been sudden and more or less violent, it is found that the cardiac nervous system has received the brunt of the toxin. Dilatation of the right heart soon becomes evident. The cardiac action grows weak and irregular. Delirium cordis sets in, and in a few days or a week the patient passes through a short agony to death. Death is usually due to heart failure, but this may be assisted by lowered tension in the peripheral circulation, or by pneumonic paresis.

*Second.*—If the onset has been less violent, and the cardiac nervous system only moderately poisoned, the case will probably run a definite course ending in complete recovery. This is especially true when a case of acute beri-beri is accorded the treatment usually meted to cases of acute toxic neuritis in an occidental hospital. Up to the tenth or fifteenth day of the disease the gastro-duodenal symptoms are readily demonstrable, and the patient complains of annoying, deep epigastric sensations. There may be a slight pyrexia now. On the other hand the disease may run its course without a rise in temperature. The preliminary pretibial œdema ceases to spread if the patient is kept at absolute rest in bed, and about the tenth day it probably disappears altogether with a bulky discharge of urine. The sensori-motor symptoms tend to extend until about the fifteenth or twentieth day, and may involve both the upper and lower limbs. The muscles first weakened become more so and decidedly flaccid, while the primary sensory disturbance develops in the legs to complete loss, and in the arms to partial loss. On the other hand, the sensory disturbance may never pass beyond hyperæsthesia and hyperalgesia, or an analgesia dolorosa alone may exist. Tenderness of the calves and arm muscles is complained of, and these parts are tender to pressure. The pulse remains soft and readily compressible. It is usually rapid, occasionally slow, and nearly always irregular. The cardiac rhythm may be of the embryonic type, with reduplication of one or several sounds, or it gallops and threatens to become delirious in character. The heart dilates and the cardiac dulness extends to the right. The apex beat is marked and yet impalpable. There is usually a marked wavy precardiac impulse which contrasts with the low, easily compressible pulse. Various hæmic bruits are heard, and there are attacks of palpitation and cardiac dyspnœa. The bases of the lungs give signs of engorgement.

This is the course and these are the signs which develop in acute beri-beri up to the fifteenth or twentieth day after the onset. The epigastric tenderness and gastro-duodenal irritation then begin to subside. In a day or two the former is no longer demonstrable, and the latter is no longer complained of. If there has been a pyrexia it disappears with the gastro-duodenal symptoms. Soon the sensori-motor disturbances begin to subside, first from the parts most recently affected, and then from

the sensory areas and muscles originally involved. Any œdema still present is now absorbed. The cardiac function approaches the normal, and by the end of the fifth or sixth week of the disease the patient is apparently whole again. The knee-jerks, however, remain absent for a year or more, and there may be a slight cardiac weakness.

The *Third* and not infrequent course of beri-beri is as follows:—Up to the third or fourth week the disease proceeds as in the second course, and there is every reason to believe that the poisoned cardiac and sensori-motor neurons are about to wholly reintegrate. But for some obvious reason, such as that the patient is allowed to get up too soon, or that he has continued on his feet during the active stage of the disease, there is a halt in the subsidence of the different paralyses and sensory disturbances. And now, what was originally an almost complete flaccid paresis, a wide disturbance of sensation, or a marked cardiac weakness, resolves into narrowly confined atrophic paralysis of the leg and arm muscles, definite anæsthesia of the legs and hands, trophic changes in and about the joints, and weakness and irritability of the heart only troublesome on exertion. What has been a general anasarca remains more or less so, or becomes reduced to a moderate œdema of the lower limbs. In time some one symptom becomes predominant. It may be cardiac weakness and irregularity, or atrophic motor paralysis, anæsthesia, analgesia, or œdema. What really happens is that certain of the originally but slightly poisoned autonomic and sensori-motor neuronal terminations fail to reintegrate on the elimination of the beri-beric toxin and now proceed to truly degenerate. This degeneration not only extends laterally in the individual fibres, but also migrates toward their trophic centres. The rapidity and extent of this fibre degeneration is not always the same in different cases. It may develop slowly, and not farther than to cause a slight atrophic weakness in the leg muscles and a slight disturbance of sensation about the ankles, or it may proceed rapidly and cause complete atrophic paralysis, marked sensory loss, voluminous œdema, or constant cardiac weakness and irregularity.

For cases of beri-beri which take this third course I have proposed the term, *Beri-beric Residual Paralysis*. This term may be modified so as to indicate which of the neuronal systems,

cardiac, motor, sensory, or vaso-motor are most markedly implicated.

Cases of Beri-beric Residual Paralysis may live for many years, depending largely on the progress of the degeneration in the terminations of the vagal cardiac fibres. The sensori-motor and vaso-motor symptoms may partially disappear. On the other hand, they may resolve to a minimum and then oscillate between this and recovery. The sensori-motor and vaso-motor symptoms never, however, penetrate to new areas, but confine themselves to those which failed to clear up on the elimination of the beri-beric toxin. Death, when it comes, is usually due to some cause inherent in the disease itself, generally cardiac failure, or to an intercurrent disease, phthisis, malaria, or dysentery.

In view of the above facts, I have classified beri-beri into—

Acute Pernicious Beri-beri, which is invariably fatal in the early acute stage of the disease.

Acute Beri-beri, which may prove fatal in the acute stage of the disease.

Subacute Beri-beri, which is never in itself fatal; and

Beri-beric Residual Paralysis, or Acute Beri-beri, and Subacute Beri-beri as they resolve and develop after the causal bacillus and its toxin have been eliminated.

#### PATHOLOGICAL ANATOMY OF ACUTE BERI-BERI.

The subjects of this form of beri-beri are in the prime of life, so that the cadaver shows no external signs of disease. If there has been a prolonged death-agony, blood-stained froth is seen on the lips. The eyes are half open and the fingers are flexed. Rigor mortis comes on quickly. On opening the abdomen the stomach and upper part of the small gut are found toneless and sometimes dilated. They are usually empty, however. The mucosa of the pylorus, duodenum, and upper part of the jejunum is markedly congested; the crests of the valvulæ conniventes and flattened rugæ are the seat of numerous hæmorrhagic injections. Patches and rings of scarlet injections occur. Malcomsen (5), who first described them, most aptly compared them to sprinklings of red pepper. From the congested mucosa one may have to strip away a thin pellicle of treacle-like blood-stained mucus, but no true membrane is ever formed. I

have observed this condition as low as the ascending colon, but in the majority of cases it is confined to the pylorus and duodenum. The first chain of mesenteric glands are found swollen and hyperæmic. There are varying amounts of clear straw-coloured fluid in the abdominal cavity. The liver and spleen are moderately swollen and engorged with blood, also the kidneys.

No other macroscopic changes are found in the abdominal organs in uncomplicated cases of acute beri-beri. If the case does not succumb until the fourth or fifth week, that is, until after the gastro-duodenal symptoms have cleared up, the congestion, etc., of the gastro-duodenal mucosa may be absent.

On opening the thorax the heart looks massive, as though greatly hypertrophied. It is but greatly dilated, however, and filled with dark, semi-fluid clot. This is especially true of the right chambers. The walls of the latter are thinned, and when emptied the whole organ collapses. There is a relative incompetence of the tricuspid valves owing to dilatation of the auriculo-ventricular ring. The walls of the right heart are sometimes spongy, and when squeezed exude blood. Fatty degeneration of the muscle may often be detected by the naked eye. The bases of the lungs are massive from hypostatic congestion and œdema. If there has been much dyspnoea and a prolonged agony, the apices and ventral borders of the lungs are emphysematous. The encephalon is normal to the naked eye. The spinal cord is usually congested in the lumbo-sacral levels. It is really remarkable that such an acute and rapidly fatal disease exhibits so few macroscopic changes.

Microscopically, however, profound changes are found in the gastro-duodenal mucosa, in the nervous system, and in the myocardium. The affected parts of the gastro-duodenal mucosa exhibit the main features of an acute inflammatory process. There is an active congestion, the lymphatics are filled with debris, and there are small extravasations into the tips of the villi without their covering epithelium being broken. The above-mentioned red pepper-like patches are seen to be composed of widely dilated adjacent capillaries. There is a marked small-celled infiltration of the mucosa and submucosa. Leucocytes are numerous in the mucosa. There are varying degrees of epithelial necrosis. It may range from a complete disintegration of the epithelium and support tissue of the villi, necks, and bodies of

the glands, to a partial necrosis of the epithelium of the necks of the glands. In some cases the necrosed villi have sloughed.

Lying in the necrosed mucosa is a bacillus of constant morphological character. It measures from 4-9  $\mu$  in length and from 1-1.05  $\mu$  in width. Its ends are slightly rounded, and it stains readily with the basic dyes. It cannot be decolourised by Gram's. All stains leave from one to three finely striated bars across the organism.

The mesenteric glands near the stomach and duodenum exhibit patches of necrosis, and are deeply engorged with red cells. No micro-organisms are present.

The muscle-fibres of the heart are found extremely fatty. This degeneration is most marked in the right walls and in the papillary muscles. Fragmentation of the muscle-fibres of the right heart is common, especially if there has been a prolonged agony with delirium cordis. A few foci of small cells are found beneath the endo- and peri-cardium. The whole of the right wall has a looser texture than normal, as though the individual fibres had been slightly dissociated. The capillaries are dilated with red cells. Many of Bidder's and Remak's ganglion cells exhibit a slight chromatolysis.

The bases of the lungs show the characteristic changes of hypostatic congestion and moderate œdema. In the kidneys a few subcapsular foci of small cells are to be seen. The epithelium of the glomerulæ and convoluted tubules is sometimes slightly granular, but more often no pathological change is visible. The capillaries are always crowded with red cells. Bar an extreme passive congestion, the liver and spleen show nothing unusual in acute beri-beri.

When we come to examine the nervous system of a fatal case of acute beri-beri, we find the true explanation of the disease. Many fibres of the different neuronal systems may be involved. To describe the changes in one system of neurons, however, is to describe them in all. The vagal, as the most important neurons affected, will do for an instance. The termination of these neurons in the heart, and as they pass through the deep cardiac plexus, show a change which is profound, but nevertheless it can only be regarded as a pseudo-degeneration. We see in fact a toxic alteration of the myelin, but not a true degeneration. The nuclei of the neurolemma and their surrounding



protoplasm are blackened by the osmic of Marchi. There are intensely black flecks at the nodes and internodes, but no rounded drops of disintegrated myelin are seen. Between the fleckings the myelin is frothy-looking, each bubble being slightly outlined by the osmic. What strikes one is that, though each individual fibre termination is only slightly changed, yet every fibre in a given nerve twig is affected. This is the serious fact in the vagal implication of acute pernicious beri-beri. It is not the slight damage to each individual termination that causes the profound cardiac disturbances, but the fact that the whole number of terminations are poisoned. In cases of acute pernicious beri-beri which do not succumb for a week or more after the onset, the pseudo-degenerative changes in the terminations of the vagal and other implicated neurons are slightly more marked and may even look degenerative. Rounded drop-lets of myelin are frequently to be seen, especially in those neurons first attacked by the beri-beric toxin.

I have invariably found that the trophic centres of all neurons affected by the beri-beric toxin exhibit chromatolysis and dislocation of nuclei. The change in the centres is always moderate in the acute stage of the disease, but it illustrates the law that the whole of a given neuron is molecularly affected when attacked in one of its parts.

In only one of my cases have I been able to find micro-organisms in the viscera after death. This case died in great agony twenty days after the onset of the disease. Post-mortem I found some erosion of the gastro-duodenal mucosa. It seems likely, therefore, that the organisms found in the viscera escaped into the circulation through the erosions. The predominant organism is morphologically indistinguishable from that described above as having been seen in the necrosed mucosa of other fatal cases of acute beri-beri. I have not made any attempt to cultivate from the tissues. But all careful attempts to cultivate organisms from the blood of cases of acute beri-beri have failed. Dr Wise of the London School of Tropical Medicine has recently examined a typical case of acute beri-beri in the seventh day of the disease, and has failed to cultivate any organisms from blood taken direct from the vein. I have no hesitation in stating that the numerous cocci, and other organisms found in the peripheral blood by Pekelharing and Winkler (6), van Eecke (7), Ger-

rard (8), Dangerfield (9), and others, are due to faulty technic, or else their cases were complicated.

#### **PATHOLOGICAL ANATOMY OF BERI-BERIC RESIDUAL PARALYSIS.**

The appearance of a cadaver of beri-beric residual paralysis depends upon the duration of the disease, and upon the extent of the degeneration in the different neuronal systems. For illustration I propose to take a case in which the motor and cardiac nervous systems have been widely affected. I will assume that there has been no intercurrent disease, and that the case has finally succumbed to heart failure. Great emaciation is present in such a case, and the muscles whose nerves are degenerated are markedly atrophic. If no rigor mortis is present the ankle, knee, and wrist joints are remarkably loose, and the knee joints permit of an extensive retroflexion. On opening the abdomen, one is at once struck by the absence of the gastro-duodenitis of the acute cases. The stomach and duodenum are not toneless and there are no signs of inflammation. They usually contain small quantities of rice or other food, for it is not infrequent for these cases to collapse from heart failure shortly after a meal. Varying amounts of clear, straw-coloured fluid are found in the abdominal cavity. If the case has been one that the older writers would have classified as wet beri-beri, the amount of fluid will be great in all the serous cavities and in the subcutaneous tissues. The liver is enlarged, and when incised looks fatty and pigmented. There is usually a slight increase in its support tissue. The spleen is also moderately enlarged and full of blood. Occasionally there is a moderate thickening of the capsule, but one has to think of malaria in such instances. The kidneys are engorged as a rule, and when cut into suggest fatty and granular degeneration. None of the other abdominal organs exhibit morbid changes. In such cases as those I am instancing, the contents of the thorax present a nearly constant appearance. The lungs exhibit both acute and chronic emphysema of the apices and ventral borders. The bases are oedematous and hypostatically congested. Often there is a moderate increase of the fibrous elements throughout the entire organs. The heart looks massive, and in contrast with the heart of an acute case it is found to be more or less hypertrophied. The hypertrophy is always of the eccentric type, and is greater on the

right than left side. The right ventricular wall is rarely thicker than 8 mm., but the cavity is greatly enlarged, and there is dilatation of the auriculo-ventricular ring. The right auricular wall is moderately hypertrophied and the cavity enlarged. The left wall has never in any of my cases exceeded 20 mm.; the average being about 18 mm. The left cavity is always moderately dilated. In about a third of my cases the right walls have been thinned to 2 or 3 mm. In any event the whole organ is flabby. This is especially noticeable when the massive, dark, semi-fluid clot is freed from the right cavities. One never sees the deep red colour and firm consistency of hypertrophied muscle. In all cases the myocardium is fatty looking, and may even show a marked pigmentary infiltration. Several of my cases have shown a mucoid atrophy of the external fat, and occasionally a fatty invasion at the apex.

Microscopically, profound degenerative changes are seen in the neuro-muscular system, and in the myocardium and its nervous system. The atrophy of the muscles is of the parenchymatous type. There is little nuclear multiplication or fibrosis. In the liver, varying degrees of fatty degeneration and brown atrophy are found. There are moderate numbers of small round cells in the portal canals and beneath the capsule. The portal connective is moderately increased, and the vessels are engorged with red cells. The epithelium of the glomerulæ and convoluted tubules of the kidneys are slightly fatty, or at times granular. A few foci of small cells are found beneath the capsule and deeper in the cortex. The changes in the abdominal viscera of beri-beric residual paralysis are never gross.

Of all the viscera, the heart alone exhibits profound modification. Fatty and sometimes brown atrophy is marked throughout the organ, but especially in the right walls and in the papillary muscles. There is often a slight increase in the intermuscular connective. A moderate number of foci of small cells are seen in the deeper tissue, but, as a rule, they are confined to beneath the membranes. The cells of Remak and Bidder are often found greatly atrophied.

In the nervous system we find that the pseudo-degeneration of the terminals of the acute stage of the disease has developed into a true atrophy, and has migrated centralwards. I will here, as in the acute cases, take the degeneration in the vagal neurons

to illustrate. On examination it is seen that the change in these neurons is no longer mild and confined to the distal terminations in the heart. It is found now to be truly degenerative, and to reach as high as the middle and even upper third of the main trunks. Large drops of altered myelin are seen along the course of individual fibres. Many fibres have completely disintegrated; no axon is to be seen, and only a few drops of myelin are left to mark the once perfect fibre. In contrast with the vagi of the acute cases we find that the degeneration is confined to a small proportion of the total number of fibres; never more than a third. It would seem, therefore, that a sufficient number of the vagal cardiac fibres recover from the pseudo-degeneration of the acute stage of the disease to carry on the cardiac function, while a minority fail to reintegrate on the elimination of the poison, but degenerating, interfere with the cardiac function, profoundly affect the nutritional stability of the myocardium, and so bring about death.

This is the simple picture of the changes found in beri-beric residual paralysis. It should be remembered, however, that degeneration of the nervi-vasorum and of vagal fibres to the lungs may be a factor in the cardiac failure which generally causes death.

#### WORKING THEORY OF THE CAUSE AND NATURE OF BERI-BERI.

On the above facts, and on many others which it is not necessary to mention in outlining the disease, I have propounded the following working theory of beri-beri.

That the disease is caused by a specific bacillus, not yet isolated, which, by contamination of food and drink, gains an entrance to the alimentary canal. That the bacillus multiplies in the contents and mucosa of the stomach and small gut, but chiefly in the contents and mucosa of the pylorus and duodenum, and there elaborates an extracellular toxin, which, gaining the circulation, poisons certain sensori-motor and autonomic neurons, to different extents and in different degrees, and thus gives rise to a group of symptoms which may be classified as acute pernicious, acute and subacute beri-beri.

That those attacked by the disease die almost at once from cardiac exhaustion, or, if properly treated, follow a definite course

ending in full recovery, or a definite course ending in narrowly confined sensori-motor and vaso-motor residual paralyses.

I would specially impress that the above outline of beri-beri is of the disease as usually seen. It should not be forgotten, however, that true relapses occur in this disease as in all other acute infections, and that one attack does not protect any more than one attack of diphtheria protects. I have several cases with a history of two and even three attacks superposed on an old residual paralysis.

Rather than blur my outline I have reserved for consideration a class of cases of beri-beri which are rare. I mean those cases of beri-beri which do undoubtedly occur in women undergoing confinement—parturition beri-beri; cases that occur shortly after surgical operation, or in old ulcer cases when warded with acute beri-berics—surgical beri-beri; and finally, such cases as those reported by Hirota (10), of beri-beri in infants, at the breast of mothers who are suffering from parturition beri-beri.

These cases, on first thought, may seem to be outside the working theory of beri-beri which I have given above. But, on the assumption that the beri-beric bacillus is one that induces its effects by the agency of an extracellular toxin, all cases in the latter categories are explained. Diphtheria furnishes an analogy, for in this disease not only does the Klebs-Loeffler bacillus induce its effects by an extracellular toxin elaborated in a mucous surface, but also in any open wound. We may safely say, therefore, that the causal organism of beri-beri is not only capable of inducing its effects in the mucous surface of the pylorus and duodenum, but also in an open vaginal or skin wound, or by the circulation of its toxin in the blood of a nursing mother.

Looking at beri-beri from all sides, it can only be explained by granting an organism which induces its effects on the nervous system through the agency of an extra-cellular toxin elaborated at the site of a primary lesion. Fortunately there is no necessity to assume so much. The large number of facts and observations which I have made are almost conclusive. It really only remains to isolate the bacillus associated with the gastro-duodenitis of acute beri-beri.

## THE PATHOLOGY OF BERI-BERI.

In the foregoing outline of the etiological, clinical, and anatomical features of beri-beri, much has necessarily been said of its pathology. Yet it will be of advantage to examine the latter aspect of the disease under a separate heading.

The logical relation of symptom to lesion is as manifest in beri-beri as in any other disease. More so, for in beri-beri no constitutional disturbance complicates, and there is only the action of a bacterial toxin on the nervous system to be considered. It is, in fact, as simple a disease as is known, if we accept the working theory outlined above.

Disregarding for the moment all irregular beri-beri, such as wound beri-beri, and confining attention to the disease as usually encountered in the Tropics, it may be said with assurance that its specific organism chooses for the site of its activity the contents and mucosa of the pylorus and duodenum, and that the gastro-duodenitis which it originates is the primary lesion of the disease.

To the activity of the specific bacillus found in this primary lesion all the symptoms of beri-beri may be traced. There is nothing to show that the symptoms of the disease are due to the extension of the bacillus within the body. We are therefore reduced to believe that it induces its effects through the agency of an extra-cellular toxin elaborated at the site of the primary lesion.

The severity and extent of the primary gastro-duodenal lesion does not bear an invariable ratio to the severity and extent of the various paralyses which supervene. In some of my cases which succumbed in from three to seven days after the onset of the gastro-duodenal syndrome, there is only a moderate necrosis and small-celled infiltration of the gastro-duodenal mucosa, and the bacilli are comparatively few. On the other hand, one of my cases with only a moderate sensori-motor paresis succumbed after three distinct relapses in the seventh week of the disease, and on autopsy exhibited an enormous number of bacilli in a markedly necrosed and inflamed mucosa. I am inclined, in view of such findings, to believe that the specific bacillus of beri-beri, when it acts in the alimentary canal, does so much in the same manner as the cholera spirillum. That is, it operates by

choice in the contents of the pylorus and duodenum and other parts of the small gut, and only invades the damaged mucosa if the cases do not at once succumb.

In regular cases of beri-beri the active stage of the bacillus appears to be short, twenty days at most. I speak now of cases which are rationally treated, and not of cases which receive no treatment at all, and die, or of cases which finally recover after several relapses. I base this view on the facts that, when cases are kept at rest in bed, there is a subsidence of the gastro-duodenal syndrome about the fifteenth or twentieth day, and that should they subsequently die in the fourth or fifth week of the disease, no evidence of the gastro-duodenitis is found. That the gastro-duodenitis has been rarely seen by workers at beri-beri is due, I think, to the fact that cases do not often die during the first few days of the disease.

It will have struck the reader that beri-beri is somewhat analogous to diphtheria. That in the former, as in the latter, we have to do with a specific bacillus which acts locally in a mucous or wound surface, and induces its remote effects through the agency of an extra-cellular toxin. In none of my sections are bacilli to be seen in healthy tissue. The necrotic mucosa alone is invaded. The earliest act of the bacillus, presumably through the agency of its toxin, is to necrose the epithelium of the villi and necks of the glands. The epithelium of the bodies of the glands is rarely necrotic, although it may stain badly. The mucous necrosis seems to be preceded by a proliferation of nuclei. A small amount of fibrin is also thrown down in the necrotic tissue, and in both the mucosa and sub-mucosa there is a more or less marked small-celled infiltration. The capillaries of the villi are swollen with red cells, and may even have ruptured and become involved in the necrotic process. There is no tendency to ulcer formation, although superficial sloughing is not uncommon. I lean to the view that an actual membrane would be formed, were it not that the peristaltic action of the gut sweeps away the most necrosed tissue and its contained bacilli directly it is formed.

It seems strange that a toxin capable of necrosing the mucosa of the pylorus and duodenum should fail, on gaining the circulation, to similarly necrose other visceral tissue. As a matter of fact, it does not do so, except occasionally the first chain of

mesenteric glands. The toxin seems to be potential only as regards nerve tissue. The likely explanation is, that having exerted itself locally on the gastro-intestinal mucosa, its necrotic power is so reduced that it causes no further recognisable necrosis.

In the act of being absorbed into the circulation the beriberic toxin attacks the nervous system. In all fatal acute cases the vagal terminations in the stomach and duodenum are found poisoned. To this may be attributed part of the epigastric syndrome which ushers in the disease. The deep epigastric pain, the subjective sensations of weight, cold, and heat in the stomach, and the relaxation and dilatation of the gastro-duodenal walls with consequent bulging of the epigastrium, etc.

On gaining the circulation the toxin acts quickly, symmetrically, and more or less widely and severely on the neurons. Certain systems of neurons never escape; others may become involved. There is no doubt that the toxin shows a special affinity for the vagal cardiac terminations. In fact the vagal terminations, both in the intestine and in the heart, are the first to suffer. Even in subacute beriberi, where there has been no demonstrable cardiac disturbance, the vagal terminations in the heart exhibit degeneration when such cases come to autopsy, as the result of an intercurrent disease. In acute pernicious beriberi the vagal cardiac terminations bear the brunt of the poison, and this, together with poisoning of the accelerator terminations, soon leads to cardiac exhaustion and death.

The twigs that subserve the knee-jerks are amongst the first to receive the impact of the toxin. The jerks are therefore almost instantly and symmetrically lost. Occasionally they pass through a few hours of exaggeration before disappearing, or, more rarely, they at once become plus, and remain so throughout the course of the disease. The first motor nerves to show the effect of the toxin are the twigs to the peroneal and anterior tibial muscles. The twigs to the recti of the legs are also early and almost invariably affected.

Of the sensory nerves, the terminations of the musculo-cutaneous in the feet and legs are the first to feel the effects of the circulating toxin. The foregoing groups of sensori-motor neurons are first, and almost invariably affected. In severer cases the action of the toxin is widespread. I have seen an



almost complete flaccid paresis of the larger number of voluntary muscles.

The œdema of beri-beri is difficult to explain. The blood changes in beri-beri are not sufficiently known to exclude them altogether as factors in the case. This is certain, that there is no kidney change sufficient to account for even part of it. Albumin is not found in the urine of uncomplicated cases, and there is never a gross lesion in the kidney structure. It would be unwise at the present juncture to ascribe the œdema to no other factor than the action of the beri-beric toxin on the nervi-vasorum. There can be no doubt, however, that the action of the beri-beric virus on the neuronal terminations in the arterioles is a potent factor of the œdema. Œdema almost invariably first appears in those subcutaneous tissues which lie between the sensory and motor areas first attacked by the beri-beric toxin. The early pretibial œdema, the loss of sensation in the front of the legs, and the loss of voluntary power in the peroneal and anterior tibial muscles are associated. This association depends upon the fact that the beri-beric toxin acts simultaneously on the neuronal terminations in the vessels, skin, and muscles of the lower third of the legs. In more extensive œdema the nervi-vasorum to the vessels in the whole œdematous area are likewise poisoned. It is probable that the action of the beri-beric toxin on the terminations of the nervi-vasorum induces a trophic disturbance in the walls of the vessels, which permits the escape of the fluid constituents of the blood.

There is then a minimum of action of the beri-beric toxin, namely: poisoning of the vagal terminations in the stomach, duodenum, and heart, of the motor twigs to the peroneal, anterior tibial, and rectus muscles of the legs and thighs, and of the twigs of the nervi-vasorum to the vessels in the lower pretibial region. The effects of the toxin may not extend beyond this minimum. On the other hand, the effect of the toxin may in a few hours involve the sensori-motor terminations of all the muscles of the limbs and trunk, the diaphragm, and even the larynx, pharynx, face, and internal and external ocular muscles. All poisoned muscles become extremely flaccid, and, from the looseness of the ankle, knee, and wrist joints, there is reason to believe that the tendon nerves have been affected.

The rapidity with which the sensori-motor disturbance

progresses is only approached by the rapidity with which it subsides on the elimination of the causal organism and its toxin. After the tenth or twentieth day of the disease, that is, shortly after the subsidence of the gastro-duodenal irritation, I have seen an almost complete flaccid paralysis and a widespread oedema and sensory disturbance subside to a minimum in a week or ten days. The explanation of this remarkable phenomenon is that the changes in the neurons is of the nature of a poisoning. As I have said above, even on the fifth or sixth day of the disease the structural alteration in the neuronal terminations is slight. The changes, in all probability, begin as an invisible histochemical reaction, just sufficient to break the synapses between the terminations and the structures to which they are distributed. The medullary sheath is probably next involved, and in a few days reacts to Marchi. Up to the twentieth day or so this reaction in the medullary sheaths cannot be said to be truly degenerative, although it may be said to indicate that a degenerative tendency has been more or less established. Given the generally good physical condition of those attacked by beri-beri, it is really not surprising that the poisonous reaction in the neuronal terminations should quickly clear up on the elimination of the causal organism and its toxin.

What has just been said is true of the majority of cases of acute beri-beri, and of the great majority of the neurons in an individual case of the disease. But there is a large number of cases which do not so rapidly or so wholly recover from the effects of the beri-beric toxin. How shall these be explained? Owing to a special susceptibility to the beri-beric toxin, to a toxin of great potentiality, or to neglect in the active stage of the disease, the poisonous impact on the neuronal terminations sets up a true degeneration in certain fibres and a residual paralysis with trophic changes results.

It is difficult to say just where acute beri-beri and beri-beric residual paralysis merge one into the other. The former really imperceptibly continues into the latter, and becomes more marked as time passes. The marked atrophic paralysis, complete anæsthesia of restricted areas, slackness of tendons and oedema of residual paralysis are due to a true degeneration which develops in the, at first, only poisoned neuronal terminations.

This outline may well be wound up with the question: Does

the beri-beric toxin act on the neurons as a whole, or only on parts of them? This question cannot be answered. All that can be said is that the peripheral terminations and the trophic centres of the neurons first and alone exhibit the effect of the poison in the active stage of the disease. It is altogether likely, however, from what we know of other neurites, and from experimental observation, that in beri-beri the entirety of each affected neuron is at least molecularly changed. In cases of beri-beri where there has been a profound poisoning of the terminations of all the sensory nerves to the lower limbs, with axonal reaction in the lumbo-sacral posterior spinal ganglion cells, there is nothing abnormal in the bulbar terminations of the same neurons. This seems evidence enough that only the peripheral terminations of the neurons are directly affected by the beri-beric poison.

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### NOTE ON A CASE OF HYPERTROPHIC NODULAR GLIOSIS.

By R. G. WHITE, M.A., B.Sc., M.B.,  
Pathologist, Glasgow District Asylum, Woodilee, Lenzie.

THIS case resembles, in many ways, one of ganglionic neuroglioma recently reported in this Journal by Dr Leonard Findlay, but differs from it in, at least, one important point, namely, the absence of ganglionic cells.

The case was that of an epileptic imbecile, æt. 17 years. There

was a history of fits since childhood. The patient died from exhaustion, practically in the status epilepticus.

There was nothing special to note in the post-mortem findings beyond the brain condition.

The brain showed two small sclerotic areas, each about one inch square. These areas stood out as pale and hard portions of the cortex. They were limited to the cortex and were present, one on the internal aspect of the left paracentral lobule, and the other at the anterior end of the right middle frontal convolution. Apart from the white glistening appearance and the firm consistency of these nodules, there was nothing to distinguish them from normal brain substance. In fact, they appeared like two frozen portions of the cortex. In Fig. 1 the sclerosed knuckle of cortex appears rather too prominent, owing to the fact that the specimen had to be tilted in order to show both cortical and cut surfaces. The pia was readily removed from the cortex generally, also from these areas: here, however, the surface had a dimpled appearance (Fig. 1). The condition seems to correspond with that described by Pellizzi and termed hypertrophic nodular gliosis or tuberosc sclerosis.

The microscopic examination of these nodules bears out the first impression that these areas are but sclerosed portions of the general cortex. There certainly are cells—few and scattered large cells—which might be taken as resembling ganglionic cells (Fig. 2), however these appear rather to be large pyramidal cells, showing more or less hyperplasia. There seems nothing to justify the supposition that the condition is a new growth. There is very marked subpial felting (Fig. 3), and the field generally of the sections from the nodules shows marked hypertrophy of the glial elements: the fibres are increased in number and size, the nuclei are large, and numerous spider-cells with well-marked processes can be made out. In the midst of the hypertrophied neuroglial elements the remains of the normal cortex can be readily seen: stunted small pyramidal cells are present. They do not form a distinct cell layer, still they appear fairly numerous, considering the amount of gliosis (Fig. 4). These cells show degenerative changes in varying degrees, some cells being represented merely by a trace of protoplasm surrounding a somewhat swollen and granular nucleus. Fig. 5 shows, for the sake of comparison, a section from a neighbouring convolution to one of the nodules. Here the cells are seen to be more numerous. They are larger and have more of the normal shape and appearance, although here also degenerative

changes are seen in the form of advanced chromatolysis and absence or stunting of the processes.

An important point in this case and one that seems to lend weight to the view that the condition is one of a localised sclerosis and not a new growth of the nature of ganglionic neuroglioma, is the fact that some of the sections show cells of a transitional nature, cells larger in size than the normal pyramidal cells found in the neighbouring convolutions, but not so large as the hyperplastic cells (Fig. 6).

There is some difficulty in explaining these hyperplastic cells. They might be considered as forms of "the inflated spheroidal cells" described by Bevan Lewis: that abnormal appearance of nerve-cells of epileptic idiots which he supposed to show signs of reversion to a simpler type and to be the physical basis of heredity. I have not, however, been able to find similar cells in the sections from the neighbouring convolutions. The hyperplasia is certainly not one of a proliferative nature, such as might be found in a new growth: there is absolutely no evidence of any karyokinetic change. It may be, however, of the nature of a degenerative change, the cells having become hypertrophied and globose at the expense of the others, which show well-marked atrophic changes.

The blood-vessels in the sclerosed areas are irregular in outline, due to hyaline change; they are congested, and are contained in dilated peri-vascular spaces. The periphery of the space is formed by a distinct felting of glial fibres, and in many instances processes from spider-cells can be seen crossing to the adventitia.

The consideration of the history of the case and of the microscopical examination, also the situation of the sclerosed areas, would seem to be in favour of the view of Pellizzi, that the condition is one of congenital abnormality, occurring at the time of formation of the tertiary sulci.

#### EXPLANATION OF PLATES.

FIG. 1.—Photograph of section of left cerebral hemisphere, showing nodule in paracentral lobule, with dimpling on its surface.

FIG. 2.—Photomicrograph of section through nodule, showing one of the so-called hyperplastic pyramidal cells. Some spider-cells are also shown, and the matting of glial fibres is well seen.

FIG. 3.—Photomicrograph (taken near the surface) of section through nodule, showing marked sub-pial felting.



Fig. 1.

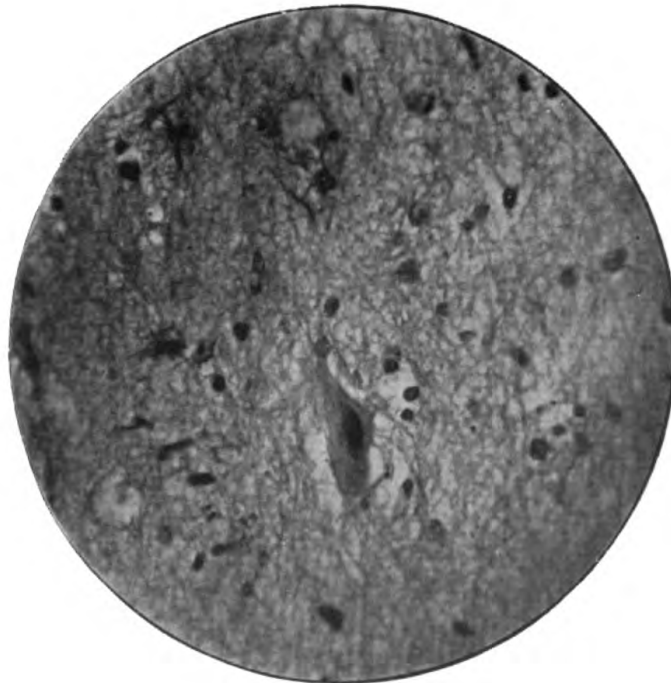


Fig. 2.



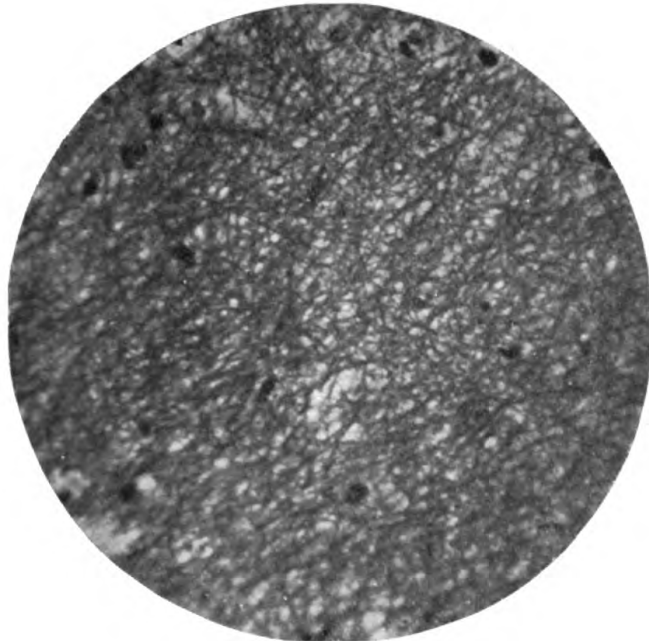


Fig. 3.

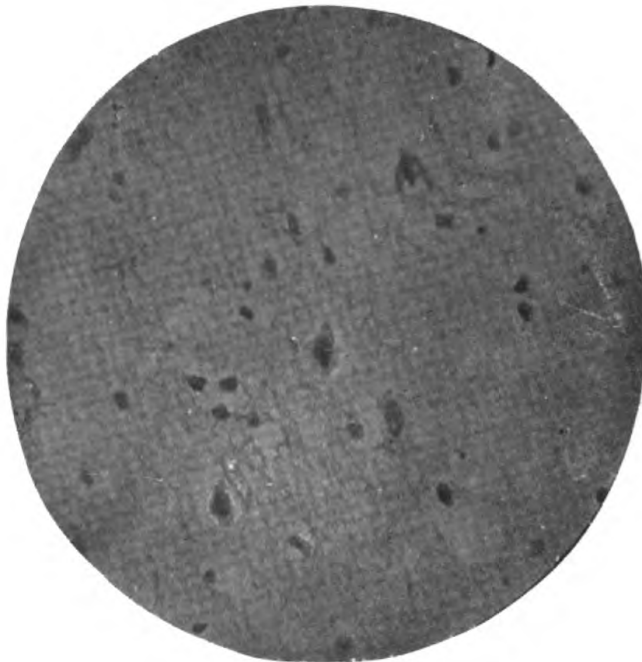


Fig. 4.





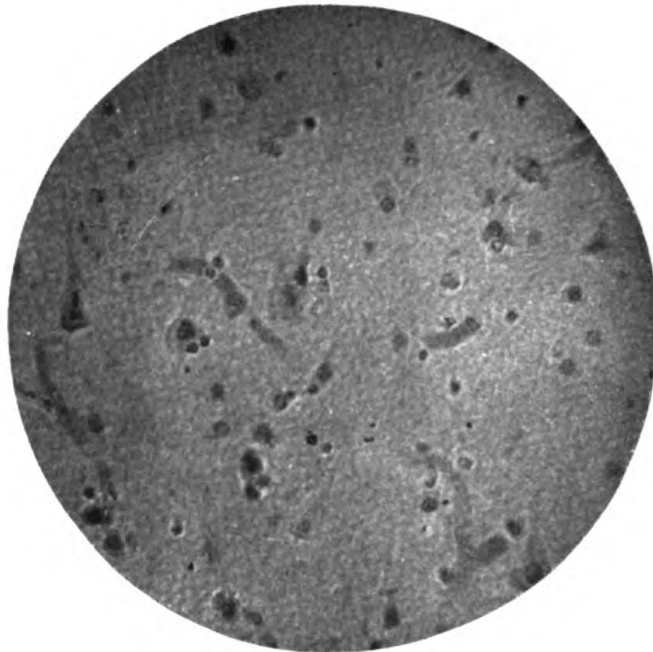


Fig. 5.



Fig. 6.



FIG. 4.—Photomicrograph of section through nodule, showing few small degenerated pyramidal cells, also increase of glial fibres.

FIG. 5.—Photomicrograph of section from neighbouring gyrus to nodule, showing larger and more numerous cells, and no glial fibres.

FIG. 6.—Photomicrograph of section through nodule, showing pyramidal cells, transitional in size between those in Figs. 4 or 5 and Fig. 2.

The microscope specimens were magnified about 200 times.

The sections were stained for the glial elements by Ford Robertson's methyl-violet method, and for the nerve-cells and blood-vessels in Toluidin Blue and Hæmatox. and Eosine.

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## Abstracts

### ANATOMY.

**ON THE STRUCTURE OF THE AXIS-CYLINDER.** (*Sulla struttura (310) del cilindrasse.*) LUGARO, *Riv. di Patol. nerv. e ment.*, F. 6, 1905, p. 265.

LUGARO recommends the following technique:—

Take the intra-dural portion of a lumbo-sacral nerve (cat, dog), fasten upon wood, and treat as follows:—

1. Fixation for 48 hours in a solution of 1 per cent. pure nitric acid in pure acetone.
2. Wash for 12-24 hours in pure acetone, changed three or four times.
3. Leave for some hours in acetone and xylol, equal parts; then in pure xylol.
4. Embed in paraffin at 50°.
5. Fasten sections (of 5  $\mu$ ) on slides with distilled water.
6. Xylol, alcohol.
7. Absolute alcohol for 24 hours.

8. Immerse for 24 hours in a solution of 1 per cent. acetic aldehyde in absolute alcohol.
9. Wash in distilled water until the alcohol is got rid of; colour by Bethe's toluidin blue method, *i.e.* about one hour in stain 1-3000; wash in distilled water; fix in ammonium molybdate; wash again in water; dehydrate; xylol; balsam.

Instead of the acetic aldehyde, formalin (watery) solution may be used, when one obtains a more intense staining reaction, but less elective. The only structures stained are the axis-cylinders and the nuclei of Schwann's sheath and of the connective tissue.

The axis-cylinders are composed of fine undulating fibrils which anastomose with each other, forming elongated lozenge-shaped reticula. Near the nodes of Ranvier, the fibrils approximate, and the whole axis-cylinder undergoes a diminution in diameter.

Possibly the neuro-fibrils are here reduced in number. In oblique section the fibrillar anastomosis is very evident.

As a result of this, Lugaro considers the whole axis-cylinder as a single physiological conductor, in opposition to the view of Bethe, who believes in separate conduction for each fibril.

Lugaro, in this connection, has not observed independent non-anastomosing fibrils, either in the cell body or in the dendrites. The axis-cylinder, with its fibrillar anastomosing network, thus presents exactly the same structure as exists in the cell body and dendrites. According to the author the fibrillar network, by the large surface which it possesses, possibly favours the process of chemical interchange, and in support of this view he mentions the observations of Cajal and Tello that in the nervous system of hibernating animals the fibrils diminish greatly in number and increase somewhat in size, while when nervous activity is increased they become more delicate.

In the fibrillar structure, therefore, there is an adaptation for both conduction and rapidity of exchange. DAVID ORR.

**CONCERNING THE POSITION IN THE PYRAMIDAL TRACT (311) OF THE FIBRES WHICH SUPPLY THE LOWER EXTREMITIES.** (Über die Lage der für die Innervation der unteren Extremitäten bestimmten Fasern der Pyramidenbahn.)  
OSKAR FISCHER, *Monatsschr. f. Psychiat. u. Neurolog.*, Bd. 17, H. 5, p. 385.

A MONOPLÉGIA of the right lower limb in an individual affected with phthisis pulmonalis. No weakness of the arm or face.

Post-mortem, a tuberculous tumour about the size of a hazel-

not implicating the cortex at the upper extremity of the left ascending frontal convolution and extending on to the mesial surface of the hemisphere.

The author concludes from the microscopical examination of Marchi preparations that the fibres which are concerned with the movements of the foot from the crus downwards are scattered over the whole of the pyramidal area and nowhere form a distinct and separate bundle.

EDWIN BRAMWELL.

### **PATHOLOGY.**

#### **AN EXPERIMENTAL STUDY ON THE REGENERATION OF (312) PERIPHERAL NERVES. S. D. LUDLUM, M.D.**

THIS paper gives the result of twenty-one experiments, mostly on rabbits, which consisted in partial or complete division of presumably the sciatic nerve, and after an attempt to secure satisfactory union, the animal was killed at periods of time extending to eight months.

Wherever there was no proper union of the divided ends, no evidence of distal axis-cylinder regeneration was found, and nothing to indicate that such regeneration does occur distally.

The author specially draws attention to his observations that in the scar tissue uniting the two ends of a divided nerve where the results were successful, axis-cylinders were well seen and were more numerous than in the peripheral end of the nerve—a fact which he holds militates against the peripheral theory of regeneration. Dr Ludlum also states that evidence is wanting of any definite union between the supposed peripherally formed axis-cylinders and the central.

Weigert's method was used for myelin staining and Stroebe's for axis-cylinders (which he states is unsatisfactory), but no description is offered of the methods of fixing or cutting the specimens. Two illustrations are given, one showing axis-cylinder regeneration from the central, and the other its absence in a distal portion of a divided nerve, thirteen weeks after section, and in which no union had occurred, but the blocks have unfortunately printed somewhat indistinctly.

Reference is made to the formidable evidence of other workers against peripheral axis-cylinder regeneration.

It is difficult for a reviewer who has espoused the peripheral regeneration theory to avoid criticism, but it is undesirable to do so where categorical statements are made.

ROBERT A. FLEMING.

**CONTRIBUTION TO THE PATHOGENESIS OF THE EXPERI-**  
**(313) MENTAL PNEUMOCOCCIC PARALYSIS.** L. PANICHI, *Riv.*  
*di Path.*, Vol. x., Fasc. 7.

THIS is an investigation into the lesions produced in the spinal cord of rabbits, sheep, and horses by injecting Fränkel's bacillus into the backs of the animals. Some of the rabbits died in about twelve hours, and in these no paralysis was observed; but in those animals which survived for some time a spastic paraplegia gradually developed.

In the sheep, a short period of asthenia was followed by a complete paralysis of three limbs. Post-mortem a peridural hæmorrhage was found, with compression and softening of the cord.

The horses suffered from a slight asthenia which soon disappeared.

In the rabbits, which died early, hæmorrhages were found in most of the internal organs. In the spinal cord the nerve cells exhibited lesions characteristic of an acute intoxication; these were accompanied by a slight change of the blood-vessels, which had in a few instances resulted in a very limited hæmorrhage. There was slight increase in the number of neuroglial nuclei.

In the animals which lived longer the nerve cell changes were less marked, but lesions of the blood-vessels were much more evident, and hæmorrhages were constantly found. The hæmorrhages were recent.

Neither the hæmorrhages nor the lesions of the medullary fibres were systematised, but were disseminated throughout the cord. Near the hæmorrhages the white matter was much rarified, the myeline sheath was gone, and the axis-cylinders were swollen irregularly.

These lesions were limited to the immediate neighbourhood of the hæmorrhages, and there were no ascending or descending degenerations.

With regard to the changes seen in the vessels, the nuclei of the capillaries were increased in number; in the veins the middle and outer coats were most affected; in the arteries the intima.

Several factors were concerned in the production of the hæmorrhages: modifications of the circulating blood, of the vaso-constrictor nerves, of the walls of the vessels, and possibly of the surrounding tissues. These might all result from the action of the toxin.

It is possible that the changes in the vessels may be due to a disturbed action of the vaso-motor centres, following the pathological changes in the nerve cells. Moreover, the vessel walls have a vitality of their own, depending on physical and chemical characters, which will be affected by the toxins.

The disappearance of the symptoms in some animals must be attributed to a re-absorption of the blood. R. G. Rows.

**A SPECIAL ALTERATION OF THE NUCLEUS IN RABIES.**

(314) L. SICILIANO, *Riv. di Patol.*, Vol. x., Fasc. 7.

THE author draws attention to the appearances of the nuclei of the nerve cells found in the cornu ammonis of rabbits which have been inoculated with rabies. With a low power, bodies are seen which are stained deeply blue with thionin. They have the appearance of shrunken nuclei, which have a great affinity for basic dyes. With a higher magnification these bodies are found to be enclosed in a membrane, and outside this there is a remnant of protoplasm.

Sometimes these bodies are round and single; at other times multiple and of different shapes.

These changes are not uniformly distributed.

Another change found is that the nuclei are small and uniformly stained with darker granules scattered throughout. The nucleolus is no longer seen. Stained with Biondi's mixture, the bodies which stain with thionin are here seen as large green drops, refractile, but showing no structure. These drops are found in the middle of a mass, little differentiated, with irregular contour, and stained red.

In the second series of bodies mentioned the granules are stained green and lie in a red mass.

These changes probably represent a regressive process occurring in the nucleus, which begins with a contraction of its body and an increase of the nuclein; this is seen either as a large drop or in the form of granules.

The author has not seen these bodies in the dog.

In the same animal it has been possible to see the lesion described, and also the bodies of Negri which lie in the protoplasm of the cell, and are stained red by Biondi's mixture.

R. G. Rows.

**THE PATHOLOGICAL ANATOMY OF EXOPHTHALMIC GOITRE.**

(315) W. G. MACCALLUM, *Johns Hopkins Hosp. Bull.*, Vol. xvi., 1905, p. 287.

THE author examined the thyroid gland from 28 cases of exophthalmic goitre. He found changes corresponding to those described by other observers. In some instances the changes were found only in foci scattered through the gland. The alveoli varied greatly in size and form. They were lined by cylindrical epithelial cells



containing fat granules. The colloid substance was diminished, and appeared in some alveoli as a shreddy granular mass, containing desquamated cells and leucocytes. Considerable bands of fibrous tissue running through the gland caused distinct lobulation of the tissue. In the interstitial tissue there was a great deal of lymphoid tissue, in the form of circumscribed, scattered lymphoid nodules. The neighbouring cervical lymphatic glands and the thymus were enlarged. The parathyroid glands were examined in nine cases and found practically normal in all.

W. T. RITCHIE.

### CLINICAL NEUROLOGY.

**FACIAL PALSY AND ITS TREATMENT.** PURVES STEWART,  
(316) *Westminster Hosp. Rep.*, 1905.

A CLEAR account is given of the causes, symptoms, course, prognosis, and treatment of facial palsy. The so-called "rheumatic" neuritis, due to cold, is not an interstitial or compression neuritis, as is usually believed, but is a primary degenerative or parenchymatous neuritis; cold is merely an exciting cause, and there must be some other factor, possibly of a toxic or infective nature.

The cause of the secondary contracture of the paralysed muscles is obscure. The common view, that it is due to a state of hyperexcitability of the cells of the facial nucleus secondary to the long-standing injury of the nerve fibres, is difficult to reconcile with the fact that it occurs only in cases of imperfect recovery, and is absent in total paralysis. Stewart suggests a different explanation: when regeneration of nerve is incomplete, the highly differentiated fibrillar element of the affected muscles is imperfectly reproduced and sarcoplasm—which is responsible for the normal "muscle tonus"—is in excess; the impulses reaching it through the regenerated nerve act constantly on the superabundant sarcoplasm and keep the muscle in a state of exaggerated tonus, *i.e.* contracture.

Electro-prognosis is of most value. The duration of the pain, which so often precedes the motor palsy, is also of some use in prognosis: the shorter the antecedent pain, the sooner is the palsy likely to pass off in most cases.

Successful results of nerve anastomosis in certain cases are recorded. On the whole, the facio-hypoglossal operation is preferable to the facio-accessory. The indications for such operation are stated thus: "A good rule is to persevere faithfully with electrical treatment and massage for six months. If, at the end of that time, no signs of recovery have appeared, we should then

recommend operation. In cases where, from traumatism, we know that the nerve has been definitely cut across, and where primary suture is impossible, we should not wait but should perform facio-hypoglossal anastomosis without delay."

ASHLEY W. MACKINTOSH.

**PARALYSIS OF ABDOMINAL MUSCLE IN POLIOMYELITIS**

(317) **ANTERIOR ACUTA OF CHILDHOOD.** (Über Bauchmuskellähmung bei Poliomyelitis anterior acuta im Kindesalter.) J. IBRAHIM und O. HERMANN, *D. Zeitschr. f. Nervenheilk.*, Bd. 29, H. 1 u. 2, 1905, p. 113.

THE authors report four cases of flaccid palsy due to acute anterior poliomyelitis in all of which there was paralysis of the transverse abdominal musculature (the recti were not involved), while in two the difference in degree on the two sides was so great as to practically constitute a unilateral phenomenon.

Oppenheim, in a paper published in 1903 which has been abstracted in a previous number of this Review (vol. i., 1903, p. 806), was of opinion that there were at that time no observations to show that a localised lesion of the spinal grey matter or anterior roots of the lower dorsal region produced an atrophy limited to a muscle segment of the rectus or obliques, but that such a lesion caused rather a diffuse weakness of the abdominal musculature of the affected side. Observations made by the authors and illustrated by photographs appear to show that a localised segment of the oblique abdominal muscles may be paralysed as a consequence of an acute anterior poliomyelitis presumably limited to the grey matter supplying the paralysed muscle segment. Similar cases described by Duchenne, Borchardt, De Quervain, and Baracz are referred to.

EDWIN BRAMWELL.

**FAMILY TABES DORSALIS: TABES IN HUSBAND, WIFE, AND**

(318) **DAUGHTER.** E. F. TREVELYAN, *Lancet*, Sept. 9, 1905, p. 755.

THE following is a very interesting sequel to the author's paper in the *Brit. Med. Journal*, 1898, on Conjugal Tabes. Of the couple there reported, the man has since died; the woman remains in about the same condition. The eldest daughter now presents well-marked signs of Tabes. In 1898 the first symptoms of the disease were already suspected in her, and they have since become more fully developed. She is now 43 years of age. Commenced having pains and occasional diplopia when 36 years old. Girdle pain and disturbance of bladder appeared shortly after. She has

now Argyll-Robertson pupil, some inco-ordination of legs, and slight Rombergism. There is a zone of hyperæsthesia about the trunk. Knee-jerk and ankle-jerk absent, plantar flexion.

Although no definite history of syphilis could be obtained in this family, and no evidences of it except what one sees in the family history, Hudovernig, in a series of cases of conjugal tabes, gives the percentage of a preceding luetic history as 96, and Raecke as 90.

Only one or two instances of tabes in husband, wife, and offspring could be found in the literature. In some cases general paralysis has taken the place of tabes in one or more of the individuals. To illustrate the importance of the relationship of syphilis to tabes and general paralysis, the cases of Erb, Finkelburg, and Wertheimer may be referred to, in which tabes or general paralysis was present in two of the individuals, and syphilis of the nervous system in the third.

Cases are fairly numerous of tabes in one parent and in the offspring. The disease usually appears in the latter at the age of five and upwards. The daughter in the author's case developed tabes at the age of 37—a late date for tabes to develop on a congenital syphilitic basis—and she had no signs of congenital syphilis except, perhaps, the tabes itself.

Quite apart from the subject of tabetic couples and their offspring, a fair number of cases are on record in which adult tabes has been attributed to congenital syphilis.

The author is of the opinion that in his cases it is not merely a question of an inherited general vulnerability of the nervous system to certain noxæ, but the transmission of the actual virus, which would bring about, with little or no further provocation, the eventual degeneration of the afferent sensory neuron.

COLIN K. RUSSEL.

**A CASE OF TUMOUR OF THE CAUDA EQUINA REMOVED  
(319) BY OPERATION, WITH REMARKS ON THE DIAGNOSIS  
AND NATURE OF LESIONS IN THAT SITUATION.**

W. B. WARRINGTON, *Lancet*, Sept. 9, 1905, p. 749.

A LAUNDRYMAN, æt. 36, had suffered for five months with pain in both thighs, and for a shorter time with weakness of legs.

*P. H.*—Four years ago had gonorrhœa and stricture. On one occasion retention of urine necessitated supra-pubic puncture.

*F. H.*—Father died of cerebral tumour.

*P. C.*—On admission he complained of severe, spontaneous shooting pains, starting about the middle line at the upper part of sacrum, and extending down the posterior surface of each

thigh and outer surface of the legs as far as the ankles. Often worse at night, and much worse with change of position, which caused it to be referred to the lower part of the sacrum. There was sharp pain on flexing the thigh on the pelvis, but no tender points of Valleix. There was no tenderness over spines, but considerable immobility of the lower part of vertebral column. Considerable loss of power in both limbs, greater on the left side. All muscles supplied by lumbo-sacral nerves were weak, but all reacted to faradism, although to a less degree on the left side. Knee-jerks brisk, right and left. Ankle-jerks absent. Plantar reflexes doubtful, sometimes flexor, at others extensor. Some loss of control of the bowels. Anal reflex fairly active. Rectum was dilated. Anæsthesia and analgesia over the region supplied by fifth lumbar and all the sacral roots.

In the course of a fortnight the paralysis and anæsthesia became more extensive, the latter spreading to inner surface of thighs and on the legs, involving to a slight degree the fourth lumbar area. The legs below the knees were slightly wasted and absolutely powerless. Very severe pain.

Operation showed an angiosarcoma involving the cauda equina. Very vascular, and only a part of it could be removed. There was marked but only temporary improvement, and recurrence three months later proved fatal, in spite of a second operation.

In diagnosis the following points are in favour of a lesion of the roots: (1) An insidious onset, with general development and progression of symptoms. Any lesion in *conus medullaris* would rapidly produce its maximum amount of damage.

(2) Pain violent in nature, spontaneous or excited by change of position.

(3) Pain over the vertebral column below the level of second lumbar spine, and rigidity of lower part of column, due to irritation of the nerves of the *dura mater*. Pain on flexing thighs on pelvis with legs extended.

(4) An asymmetrical development is a radicular symptom, and is perhaps especially suggestive of an extra-dural lesion.

Remission of symptoms, especially amelioration of the condition of the rectum and bladder and the slow appearance of wasting, of the reaction of degeneration and of trophic disturbances, also point to the cauda equina.

As to the nature of the lesion in cauda: Dufour, in an analysis of 100 cases, found that those due to trauma were most common; next came tumours, of which he collected 21 cases; then hydatid cyst, of which there were 8, all occurring in women; and lastly, meningitis and neuritis, which is very rare.

COLIN K. RUSSEL.

**THE SIGNIFICANCE OF THE EYE-SYMPTOMS IN DIS-**  
**(320) SEMINATED SCLEROSIS OF THE BRAIN AND SPINAL**  
**CORD. W. UHTOFF, *The Ophthalmoscope*, Sept. 1, 1905, p. 429.**

PROFESSOR UHTOFF in this paper summarises his experience of the eye-symptoms of disseminated sclerosis. He does not touch upon the literature or history of the subject, but refers his readers for further details to his previous well-known contributions.<sup>1</sup> The present remarks constitute a résumé based on 150 personal observations. Atrophic alterations in the optic discs are met with in nearly half the cases. He divides the cases which present these changes into three groups:—

1. Atrophic discoloration of the whole papilla.
2. Incomplete atrophic pallor of the whole papilla, so that the inner parts of the papilla still retain a slight reddish tint.
3. Partial limited atrophic discoloration of the temporal part of the papilla.

In 5 per cent. of his cases, optic neuritis was noted, and in a similar percentage disturbances of vision occurred with no diseased appearances on ophthalmoscopic examination.

The author classifies the anomalies of the visual fields which occur in disseminated sclerosis into six groups:—

1. In one-half of all cases with anomalies of the visual fields, central scotomata, usually bilateral, often relative, with no alteration of the periphery of the field are present.
2. In rare cases peripheral contraction of the field occurs together with scotomata.
3. In a considerable percentage of cases there is an irregular peripheral contraction of the field.
4. Isolated cases present regular concentric contraction. This condition occurs in pure multiple sclerosis as well as in those cases which are complicated by hysteria.
5. Uhtoff has seen one case in which there was a ring scotoma, and another in which
6. A central scotoma was present, but cleared up, and then a persistent peripheral contraction of the field appeared.

The author has only seen one case in which there was permanent bilateral complete blindness. Charcot, he remarks, never met with this condition in multiple sclerosis.

Anatomical examination of the optic papilla shows not uncommonly a simple atrophic process spreading from a sclerotic area; in other cases pronounced interstitial changes of an inflammatory character.

<sup>1</sup> *Arch. f. Psychiat. u. Nervenk.*, xxi., 1 u. 2, 1899; "Handbuch d. Augenheilk.," 2 Aufl., Bd. xi., Kap. xxii., 11, 1904.

Pronounced paralysis of the ocular muscles occurred in 20 per cent. of the cases. If the slighter degrees without diplopia had been included, the percentage would have been much greater.

The sixth nerve is comparatively most often affected—the third nerve rarely, and only partially. Complete paralysis of all the orbital muscles Uhtoff has only twice seen. In one of these cases in which there was an autopsy, extensive sclerotic changes in the floor of the ventricles and aqueduct of Sylvius were shown to be present.

Actual nystagmus occurred in 12 per cent. of his cases; nystagmus-like twitchings in 46 per cent.; in 16 per cent. in a lateral; in 30 per cent. in all directions. Uhtoff has never met with a convergent and divergent nystagmus in this disease.

Loss of the pupil light reflex and miosis occurred in 0.75 per cent., and likewise unilateral mydriasis with abolition of the light reflex; miosis with very slight light reaction was present in 5 per cent. In the case of loss of the pupil light reflex, the autopsy showed extensive sclerotic changes in the floor of the fourth ventricle and aqueduct of Sylvius as high up as the third ventricle.

An abnormally active pupil light reflex, also hippus, have no diagnostic significance.

EDWIN BRAMWELL.

**TYPHOID MENINGITIS.** RUFUS I. COLE, *Johns Hopkins Hospital* (321) *Reports*, Vol. xii., 1904, p. 379.

DR COLE classifies cases reported as typhoid meningitis into three groups.

GROUP I. Cases showing symptoms of meningitis in which no meningeal lesions were demonstrated, or in which no definite relationship between the bacteria and the symptoms were found. *Meningisme*.

GROUP II. Cases showing symptoms of meningitis in which the relationship of the *bacillus typhosus* was demonstrated by its cultivation from the cerebro-spinal fluid during life or at autopsy, and in which the demonstrable lesions were not of a suppurative character. Cases of so-called *serous meningitis*.

GROUP III. Cases of *purulent meningitis*. It is probable that with increased knowledge many of the cases described as *ménin-gisme* will be included in the second group, in which the changes are mainly microscopical, consisting principally of œdema and round-celled infiltration about the blood-vessels, which are frequently congested. Where typhoid bacilli are present in the spinal fluid, the chances are that there is at least a beginning of a true inflammatory reaction; therefore, in their presence, it seems

justifiable to speak of a meningitis. In eight cases typhoid bacilli were cultivated from the cerebro-spinal fluid, though in only one of these were they found on microscopic examination. Since in some of these cases recovery took place, it does not follow that the serous meningitis is simply an early stage of the suppurative form, the development of which is prevented by the early death of the patient.

In most cases in which meningitis was found, the diagnosis was fairly evident; in some, indeed, the meningeal symptoms were so prominent as to make the diagnosis of typhoid fever difficult. There are no special distinctive clinical features which distinguish a typhoid meningitis from, for instance, a general acute meningitis such as that which is due to the pneumococcus.

It is not possible from the clinical symptoms to distinguish the three groups above mentioned from each other. Optic neuritis and a well-marked Kernig's sign may occur in cases of *méningisme*, while the latter sign may be absent even in the most severe cases of typhoid meningitis.

In performing lumbar puncture, cultures should be made from large amounts, 4 to 6 c.c. of the fluid. Possibly lumbar puncture may be of therapeutic value in preventing a serous meningitis from becoming purulent.

Abstracts of a large number of reported cases of typhoid meningitis are presented.

EDWIN BRAMWELL.

**NOTES ON CEREBRAL LOCALISATION.** ASHLEY W. MACK-  
(322) INTOSH, *Scot. Med. and Surg. Journ.*, Sept. 1905, p. 210.

THE author presents a summary of the most recent views which are held regarding certain questions of cerebral localisation.

Experimental, clinical, embryological, and histological evidence is accumulating to show that in man and the higher apes the cortical motor areas are situated entirely in front of the fissure of Rolando.

A separate location of sensation in the cortex distinct from the motor centres is the view held by Ferrier, Mills, and others. Dana, Horsley, Starr, Dejerine, and an increasing number of observers maintain on the contrary that the motor centres are also sensory. The arguments advanced by both sides in this discussion are detailed.

Paralysis of voluntary motor action dependent upon lesions which do not involve the motor cortex or pyramidal tracts is next considered. In this connection, mention is made of the motor palsy produced by Mott and Sherrington in the ape by dividing posterior spinal roots, and of the cases of "Soul Paralysis" recorded by Bruns and Hoppe. The former case was that of "a

man who, after an apoplectiform attack, showed *inter alia* right-sided diminution of senses of touch and pain, muscular sense, and sense of posture. The right arm was never used spontaneously, but lay as if paralysed alongside the patient; the arm was, however, used for reflex acts, *e.g.* scratching, and, when the patient was shown how to make a movement, and especially when the movement was made passively on the arm several times, he was able to repeat it perfectly. Post-mortem the motor areas were quite intact; there was softening of the cortex and sub-cortex, of the temporal, parietal, and angular convolutions." Mackintosh refers to a personal observation which he believes to have been an example of this condition.

The remainder of the paper is taken up by a consideration of the stereognostic sense, which is to be regarded rather as a judgment or faculty than a simple sense. Recent analyses tend to show that the chief factors are the spacing sense (*i.e.* the ability to distinguish two or more impressions made at one and the same time), the localisation sense, and the muscular sense, especially the position of the fingers. The author holds that the impressions got from handling an object must call into action the visual centres before the object is recognised, but it must be admitted that the relative importance of the different factors in the concept of an object may be altered by special circumstances, *e.g.* a congenitally blind person may recognise objects by handling, although there are no visual impressions at all.

A case of brain tumour is described in which there was combined tactile and optic aphasia, *i.e.* the patient could recognise most objects equally well by sight and handling, and could indicate their uses, but could not name them. The tumour was located in the left lower occipito-temporal region, and removed by operation. Vörster has collected nine cases of tactile aphasia: in every one there was also optic aphasia. Mackintosh is of opinion that his case, 'while it may not *prove* anything, seems to afford some evidence of the view that stereognostic perception alone is not sufficient, but requires the activity of the visual centres, at least before an object can be named by handling."

EDWIN BRAMWELL.

#### **SYMPTOMS OF DISEASE OF THE TEGMENTUM OF THE (323) CRURA CEREBRI. (Syndrome de la Calotte Pedonculaire.)**

E. GRUNER and M. BERTOLOTTI, *Nouv. Icon. de la Salpêtrière*, mars-avril 1905, p. 159.

IN this article the authors describe two cases of lesion in the tegmentum of the crura.

The first patient was a man, 28 years of age, who after a fall on the head developed headache and some ocular difficulty. On



admission, he was found to have a slight right-sided hemiplegia, with intention tremor in the right hand and scanning speech. There was some loss of stereognostic sense in the right side. On examining the eyes he was found to have double ptosis, slight double exophthalmos, absolute loss of upward and downward movements of both eyes, and inability to converge. There was slight divergent strabismus, and some nystagmus on lateral deviation. The fundi showed choked discs on both sides.

There was no improvement under a course of iodide and mercury, and the patient left hospital with the condition unchanged.

The second patient was a man of 22, with a tubercular history. Two years before admission he had an attack of double vision. This was followed by double ptosis, headaches, and right-sided weakness.

On examination it was found that there was comparatively little motor weakness, but much right-sided inco-ordination. There was loss of acuity of all forms of sensation down the right side, complete in extent, but only partial in degree. Examination of the eyes showed double ptosis, inability to look upwards or downwards or to converge, whilst lateral movements were good. There was divergent strabismus. The light reflex and the accommodation reflex were abolished.

There was bilateral concentric contraction of the visual fields, but no hemianopsia. Optic neuritis was present on both sides.

Later symptoms which became manifest in hospital were vomiting, keratitis in the left eye, polyuria, pains in the right arm, and spasmodic movements of the right arm and leg.

At the autopsy there was found extensive tuberculosis of both lungs. A tubercular mass was found in the upper part of the pons Varolii, surrounding the aqueduct of Sylvius, and involving the whole of the grey matter surrounding the aqueduct, including both third nuclei. On the left of the tubercle was a zone of softening destroying the inner and upper half of the fillet, and involving the left anterior quadrigeminal body. The superior cerebellar peduncles were not affected, neither were the red nuclei involved.

The authors discuss whether these symptoms are due to nuclear or supra-nuclear lesions, and quote many other cases in support of the views advanced, concluding that the nuclear and not the quadrigeminal lesion is the essential one. They are of opinion, from their microscopic examination, that the fibres from the sixth nucleus to the opposite internal rectus enter the third nerve without relay.

Clinical and anatomical illustrations are given to indicate the main features of the cases.

STANLEY BARNES.

**ON THE PATHOGENIC MECHANISM OF CONVULSIVE**  
(324) **LAUGHING AND WEeping, AND ON THE MOTOR**  
**FUNCTIONS OF THE LENTICULAR NUCLEUS.** (Sul  
meccanismo patogenetico del riso e del pianto spastico e  
sulla funzione motoria del nucleo lenticolare.) F. FRANCESCHI,  
*Riv. di patol. nerv. e ment.*, 1905, p. 249.

FOR a long time clinicians have observed that certain cases of cerebral lesions, unable to execute normal voluntary movements with the facial muscles, are still able to smile or weep emotionally. Such a syndrome is generally regarded as characteristic of a lesion of the optic thalamus, as has been emphasised specially by Nothnagel. Moreover, Bechterew has made a number of interesting experiments bearing on this point. Removal of the cerebral hemispheres, down to (but not including) the optic thalami, in amphibians, birds, and mammals, renders them incapable of performing spontaneous facial movements, either voluntary or emotional. But emotional movements can be elicited reflexly with a constancy unattainable in normal animals. On the other hand, if the thalami be extirpated, emotional movements are no longer produced, even reflexly. If, again, the thalami be destroyed, the cortex remaining uninjured, these animals can perform voluntary movements, but are unable to express their feelings by mimetic expression. Even the most painful stimuli merely produce, at most, a cry or general restlessness. Clinically, Bechterew explains the occurrence of forced smiling and weeping (the grimace of weeping) as due to lesion of the cortico-thalamic fibres. If these fibres are paralysed, the mimetic centre reacts more or less convulsively to every stimulus, whether from the periphery or from the cortex by some other route. Brissaud, on the other hand, whilst in the main agreeing with Bechterew's hypothesis, localises the cortico-thalamic path in the anterior segment of the internal capsule, more especially in its lower division, or Arnold's bundle. If, says Brissaud, the anterior part of the internal capsule be destroyed, the patient can no longer stimulate the mimetic centre for the opposite side of the face, and, therefore, smiles only on one side. If the lesion involves the small geniculate bundle (the voluntary tract for the face), the patient loses the power of voluntary facial movement, but retains emotional movements. If by a bilateral lesion both geniculate bundles are interrupted, voluntary movements of the face are lost, whilst all the stimuli from the cortex, crossing the anterior part of the internal capsule, reach the mimetic centre, and set in motion the bulbar nuclei in an excessive, unregulated fashion. According to Brissaud, this syndrome occurs whenever the cortico-bulbar paths are bilaterally destroyed in the geniculate bundles, but it may also be produced by a bilateral irritative lesion of the

cortico-thalamic paths in the anterior limb of the internal capsule.

Mingazzini also supports the theory of the mimetic function of the optic thalamus, and states that this centre is controlled by stimuli from the periphery and also by psychical stimuli from the cortex. Peripheral stimuli are transmitted by senso-thalamic neurones, cortical stimuli by psycho-thalamic. Pathological investigations would indicate that such paths pass through the corpus striatum, probably through the putamen. Other thalamo-bulbar paths running in the tegmental region of the pons would connect the thalamic mimetic centre with the facial nuclei in the bulb, whilst finally cortico-bulbar paths join the cortical face centre with the facial nucleus in the bulb, passing through the genu and the pes pedunculi. According to Mingazzini, forced or spastic weeping and smiling are the result of a lesion, either of the psycho-thalamic paths, or of the cortical face-centre, or of the voluntary cortico-bulbar paths from the face-centre. Only in exceptional cases does he admit the possibility of the causal lesion being one irritating the thalamic centre, and at the same time paralysing the cortico-bulbar paths.

Franceschi's own case was that of a woman of 46, who, eleven years previously, had an attack of right hemiplegia, with some affection of speech, both of which, however, cleared up to a considerable extent, leaving the patient somewhat emotional. Six years later she had a second apoplectic attack, followed by complete loss of speech, increase in the right-sided hemiplegia, and a well-marked, left-sided hemi-paresis. The only word she could utter was "mamma." Deglutition was impaired after the second attack, and later the sphincters became uncontrolled.

The patient was unable to speak. She made attempts to do so, which only resulted in guttural, inarticulate noises. To these was frequently superadded a paroxysm of spastic weeping, affecting both halves of the face, the right more than the left. More rarely she showed a spasmodic smile. Her attention and perception of surrounding objects seemed acute. She had never learned to read or write. She was excessively emotional, crying whenever spoken to. Voluntary facial movements were markedly weak; she was unable to whistle, blow out the cheeks, or show the teeth. Mastication and deglutition were also much impaired. She had no control over the sphincters. Death occurred from marasmus.

On pathological examination, two foci of old softening were found, one on each side. That on the right side implicated nearly the whole of the anterior segment and genu of the internal capsule, the putamen, globus pallidus, caudate nucleus, claustrum, and external capsule. The posterior part of the internal capsule was scarcely affected, and only the extreme outer part of the

thalamus. The other focus, on the left side, destroyed only the posterior half of the putamen and a very small portion of the posterior segment of the internal capsule.

From these results Franceschi adheres to the generally accepted view which regards the optic thalamus as the co-ordinating centre for mimetic movements. He then proceeds to discuss the probable course of the cortico-thalamic fibres and the effects of injury to them. He rejects Brissaud's view, which localises them in the anterior part of the internal capsule, and adheres to Mingazzini's opinion, placing the psycho-thalamic path as running through the putamen. A lesion of this path will cause spasmodic weeping and smiling. He does not consider that there are special distinct paths for smiling and for weeping. The greater frequency of weeping over smiling depends on the degree of mental depression which is habitually present.

The loss of speech in his case he regards as pseudo-bulbar, an anarthria rather than a real aphasia. He believes that the putamen has an important function in transmitting fibres for articulation. Nor is the function of the lenticular nucleus confined to a mere co-operation in the production of motor speech. It probably gives origin to, or at least transmits, motor fibres which, associated with the pyramidal fibres running in the internal capsule, go to the cord and supply the limbs of the opposite side. The motor function of the lenticular nucleus thus supplements that of the posterior part of the internal capsule. Thus in his patient, where the posterior part of the internal capsule was practically intact, there was severe right hemiplegia and left hemiparesis; moreover, it was only after the second attack that the right hemiplegia attained its degree of severity, the result of the left-sided lesion. The motor function of the lenticular nucleus is thereby rendered highly probable.

PURVES STEWART.

**BRAIN TUMOURS; A STUDY OF CLINICAL AND POST-  
(325) MORTEM RECORDS BEARING ON THEIR OPERABILITY  
AND THEIR SYMPTOMATOLOGY.** G. L. WALTON and  
W. E. PAUL, *Journ. Nerv. and Ment. Dis.*, Aug. 1905, p. 481.

THE estimates of different writers as regards the percentage of operable brain-tumours have ranged widely, *e.g.* from 3 per cent. (Seidel) to 17 per cent. (Dana), the average being about 11 per cent. From analysis of autopsy records of 424 cases, Walton and Paul find that 30, or 7 per cent., were operable; 338, or 80 per cent., were inoperable; 56, or 13 per cent., were placed in the doubtful class. Excluding cases without diagnostic symptoms and those with metastasis or infection elsewhere, they find the operable

cases reduced to 14, or 3·3 per cent., the doubtful to 34, or 8 per cent.

Some practical points were brought out by their study:—  
(1) Where a tumour is not found at the spot trephined, it is wiser to explore over the surface of the brain than to puncture the brain substance in various directions. (2) Whereas the clinical diagnosis of gumma is frequently made, this diagnosis is rare in recent post-mortem reports: the clinical diagnosis of gumma should, therefore, not deter from operation. (3) It is important to follow a fixed plan of operation in spite of previous failures: in cases with practically identical symptoms, operation may fail to disclose the tumour in one case and succeed in the next.

Certain clinical features of cerebral tumour are emphasised. Convulsions in themselves afford little or no help in locating the tumour: convulsions were found to accompany tumours in every situation. Little assistance in localisation is to be expected from the pupillary disturbances. As regards reflexes, the evidence of the present series of cases is "to strengthen the views of those who regard the brain not simply as inhibitive, but as playing an active (and, in health, the principal) part in the production of the so-called reflex phenomena." The superficial reflexes were diminished in 12 out of 21 cases (in 7, only on the paralysed side). In 135 cases, the knee-jerks were diminished or lost in 32 (in 5, only on the paralysed side), increased in 53 (on the side of the tumour only, in 1; on the opposite side only, in 13), normal in 50 cases. The diminution of the superficial reflexes in more than one-half of the cases in which these reflexes were noted and the greater percentage of cases with increased knee-jerk (41 per cent.) as compared with cases with diminished knee-jerk (25 per cent.) are believed to bear out the supposition of Grasset that, in man, the controlling centre for the superficial reflexes has risen to the cortex, while the controlling mechanism of the deep reflexes has ascended only to the region of the basal ganglia (particularly the red nucleus, van Gehuchten's tendon-reflex centre). This supposition is also supported by cases of tumour limited to the cortex, in which the deep reflexes were exaggerated, while all the superficial reflexes were diminished or lost on the paralysed side and normal on the other.

Discussing shortly the view that diminished knee-jerks in cases of central tumour are due to posterior column degeneration or, at least, to damage of the spinal cord, the writers do not believe that any such theory, which accepts the spinal cord as the sole seat of the reflex arcs in man, can explain all the facts brought out by their cases. Thus it is difficult, on any such view, to explain cases in which the reflex is lessened only on the side opposite the tumour, or to account for the not infrequent occurrence of

temporary loss of knee-jerk on the paralysed side in the early history of cerebral hæmorrhage.

ASHLEY W. MACKINTOSH.

**THE DIFFERENTIAL DIAGNOSIS BETWEEN CEREBELLAR (326) TUMOUR AND CHRONIC HYDROCEPHALUS. (TOGETHER WITH A CONTRIBUTION TO ANGIOMA OF THE CENTRAL NERVOUS SYSTEM.)** [Zur differential Diagnose zwischen Kleinhirntumoren und chronischem Hydrocephalus. (Zugleich ein Beitrag zur Kenntnis der Angiome des Zentralnervensystems.)] RUDOLF FINKELNBURG, *D. Zeitschr. f. Nervenheilk.*, Bd. 29, H. 1 u. 2, 1905, p. 135.

PROBABLY the majority of cases of chronic hydrocephalus in the adult are diagnosed as intracranial tumour. The distinction between chronic hydrocephalus and tumours situated in the cerebellum or third ventricle may be very difficult, and in some instances in the present state of our knowledge is probably impossible. It is only by collecting cases of this kind which have come to autopsy that advancement in diagnosis is to be expected. Dr Finkelnburg reports three cases in this paper.

CASE I. A boy of fourteen who had been ill for  $2\frac{1}{4}$  years with headache, giddiness, unsteady gait, diplopia, difficulty in passing urine, paræsthesia in the back and extremities. Later, defective memory, irritative phenomena, pain at the back of the head and neck. On examination, two years after the commencement of his illness, double optic neuritis and paralysis of the left sixth nerve were found; the reflexes were normal.

The diagnosis arrived at was a tumour of the left posterior fossa, although an acquired chronic hydrocephalus could not be excluded with certainty.

An operation was performed, death following shortly after.

At the sectio a moderate degree of hydrocephalus with pushing forward of the infundibulum and pressure on the optic and abducent nerves. On the floor of the fourth ventricle, which was not dilated, a small cavernous angioma resting upon the corpora quadrigemina was found, together with chronic changes in the choroid plexuses.

CASE II. A girl, aged seven years, who had been ill for seven months with vomiting and frontal headache. Three months later, unsteady gait and diminution of vision developed.

Examination, five months after the commencement of her illness, showed indistinctness of the edge of both optic discs, great defect of vision, tenderness over the left posterior fossa, unsteady

gait, ataxia preponderating in the right arm and leg, incontinence of urine and fæces. Tendon-jerks and skin reflexes normal.

Ten days later, much greater defect of vision, inability to stand or walk, the knee-jerks scarcely obtainable, somewhat indefinite extensor responses; very marked ataxia in the right arm and leg, less in the left.

A cerebral tumour was diagnosed and an operation over the right lobe of the cerebellum was undertaken. Nothing was found and the patient died on the fifth day after the operation. At the post-mortem a moderate degree of hydrocephalus was discovered; the choroid plexuses and membranes showed no macro- or microscopical changes.

CASE III. Mrs E., aged thirty-six, was examined four weeks after the commencement of her illness, which had begun with acute vertical headache and vomiting. Three weeks after these symptoms appeared, unsteady gait, giddy attacks and defective sight developed.

When examined she was found to be slightly stupid; there was marked optic neuritis in the left eye, only slight in the right, distinct tenderness on percussion over the vertex, pronounced staggering gait, tendon jerks brisk, alteration in the pulse on changing position, Schmidt's symptom positive.

A tumour of the cerebellum was diagnosed.

Six weeks after the appearance of the first symptom she died suddenly after a large quantity of fluid had discharged through the nose.

Post-mortem a tumour of the right corpus striatum was discovered.

Conclusions arrived at from these observations are:—

1. A cerebellar gait may appear as an early symptom in a case of chronic hydrocephalus or in a tumour of the central ganglia.

2. A normal condition of the tendon reflexes and even a diminution of the same does not exclude chronic hydrocephalus.

3. Schmidt's symptom is not pathognomonic of cerebellar tumours, but may also be found in cerebral growths.

4. Localised tenderness on pressure or percussion of the skull may be present in a case of chronic hydrocephalus and is only of slight value as a localising symptom.

5. More intense optic neuritis on one side than the other does not indicate with certainty that the tumour is on the same side.

EDWIN BRAMWELL.

**CONTRIBUTIONS TO THE DIAGNOSIS OF BRAIN TUMOUR**  
 (327) **AND SEROUS MENINGITIS.** (*Beiträge zur Diagnostik des*  
*Tumor Cerebri und der Meningitis Serosa.*) H. OPPENHEIM,  
*Monatsschr. f. Psychiat. u. Neurol.*, Bd. xviii., H. 2, p. 135.

THIS paper consists in the detailed records of nine cases of brain tumour and two of internal hydrocephalus, with remarks appended to each report. The cases are particularly instructive as showing some of the difficulties of diagnosis, since the ante-mortem is fully given and discussed in the light of the results found on operation or post-mortem.

CASE 1. Gliosarcoma of pons at level of sixth and seventh nuclei. Diagnosis confirmed by autopsy.

CASE 2. *Diagnosis.*—Tumour in the posterior fossa (within the vermis of the cerebellum or the ponto-medullary-cerebello-peduncular region), very probably tuberculous. No operation. *Autopsy.*—Two tuberculous tumours situated almost symmetrically on opposite sides of the medulla.

CASE 3. *Probable Diagnosis.*—Cysticercus on the floor of the fourth ventricle. No operation. *Autopsy.*—Cysticercus on the floor of the fourth ventricle, and in addition a few small calcified cysts in the cerebral cortex.

CASE 4. *Probable Diagnosis.*—Tumour of the motor area. *Operation.*—Glioma found in upper part of ascending frontal convolution. Death a few hours after operation.

CASE 5. *Diagnosis.*—Brain tumour in the upper half of the right central region. *Operation.*—Angiomatous tissue exposed, extirpation not possible. Death five weeks later. *Autopsy.*—Diffuse new growth in the region of the central convolutions and the adjacent frontal lobes, additional nodules on the mesial wall of the hemisphere at the level of the gyrus fornicatus.

CASE 6. *Diagnosis.*—Tumour in the right parietal lobe or in the right internal capsule, the first the more probable. *Operation.*—Sub-cortical tumour in the above-mentioned situation. Partial removal. Death three months later.

CASE 7. *Diagnosis.*—Cerebral tumour in the region of the left central ganglia, below the insula, the first temporo-sphenoidal convolution, very probably the lenticular nucleus. *Operation.*—Cyst with resistance in the region below the first temporo-sphenoidal convolution. Cyst evacuated. Death a few months later. No autopsy.

CASE 8.—*Diagnosis.*—Sub-cortical tumour of the right frontal lobe or corpus striatum. *Operation.*—Small trephine hole made and ventricle punctured. Death six months later. *Autopsy.*—Tumour on the surface of the right frontal lobe, cyst lying in the white matter of the right hemisphere.

CASE 9. *Diagnosis.*—At first lay between a cerebellar tumour



and a tumour of the left frontal lobe, later definitely diagnosed as a tumour of the left cerebellar hemisphere. No operation. *Autopsy*.—Tumour in lower part of left cerebellar hemisphere.

CASE 10. *Probable Diagnosis*.—Tumour in the left posterior fossa, perhaps a neurofibroma of the auditory nerve, but diagnosis uncertain, particularly the exclusion of serous meningitis. *Operation*.—Cerebellum explored with a negative result. *Autopsy*.—Chronic internal hydrocephalus.

The patient was a boy æt. 12. Examined on February 4, 1904. Tuberculous family history, otherwise nothing of etiological importance. Three years previously he had an attack of fever, vomiting, and pain in the neck. After this he remained well until October 1903, when vomiting and giddiness developed; from that time these symptoms, together with headache, persisted.

*State*.—Bilateral post-neuritic atrophy of the optic nerves with almost complete blindness on the left side, and marked diminution of visual acuity on the right. Peculiar position of the head, slight contracture of the left sterno-mastoid and trapezius. Eyes frequently directed towards the left, but freely movable. Slight nystagmus on extreme lateral deviation. Tinnitus in left ear. Tenderness on pressure and on percussion, particularly over the left occipital region. Slight cerebellar ataxia. Paresis of the right lower face. Right leg somewhat weaker than left. Indefinite Babinski sign on the right side. Right knee-jerk rather greater than left.

CASE 11. *Diagnosis*.—Serous meningitis ultimately associated with encephalitis. *Autopsy*.—Internal hydrocephalus. Symmetrical areas of softening in the cerebellum, which do not present, however, microscopically the appearance of softening.

A girl, aged 19, was first examined in April 1904. After confinement in May 1903, she often felt chilly and "out of sorts." Later, much headache, vomiting, and giddiness. A few months ago she was unconscious for five days, swelling of the legs, prominence of the eyes, and weakness of right side of body. After this repeated attacks of loss of consciousness of short duration, with convulsions. For a few weeks failing sight, complains of severe headache, frequent vomiting, intercostal pain, and pain extending up the back to the occiput.

*State*.—Double optic neuritis with appearances of albuminuric retinitis, but no signs of nephritis. Bilateral amaurosis about equal on the two sides. Slight right hemiparesis; later the left leg was weaker than the right. Tendon reflexes diminished, and later disappeared completely. Tinnitus in right ear. Tenderness on percussion all over the skull, sometimes the left side particularly painful. Percussion note "*klirrend*." Pressure on spinous processes and vertebræ painful.

EDWIN BRAMWELL.

**PROFESSIONAL CRAMP, AND ITS TREATMENT BY MASSAGE**  
 (328) **AND RE-EDUCATION.** (*La crampe professionnelle et son traitement par le massage méthodique et la rééducation.*)  
 P. KOUINDJY, *Nouv. Icon. de la Salpêtrière*, mars-avril 1905,  
 p. 215.

A REVIEW is first given of the history of this subject, particularly as to the etiology and the treatment which have been adopted by former observers. The author compares professional cramp with locomotor ataxia, suggesting that in both conditions we may have an ataxic, a paralytic, a spasmodic, or a tremulous form. "Furthermore, we find in professional ataxia a psychic element, which is one of the characteristics of locomotor ataxia." Heredity, gout, rheumatism, trauma, cold, the use of metallic pens, and bad attitude in writing are discussed as causes of the disease.

After a description of the various types of the disease, the author discusses the means of treatment which have been adopted. The treatment he adopts consists of re-education combined with a systematic massage, calculated to strengthen those muscles which are relatively weakened, particularly those of the extensors of the forearm. Diagrams are given to illustrate the exercises which are recommended.

STANLEY BARNES.

**ON THE INTERPRETATION AND IMPORTANCE OF APHASIC**  
 (329) **DISORDERS IN EPILEPTICS.** (*Ueber die Auffassung und Bedeutung aphasischer Störungen bei Epileptikern.*)  
 K. HEILBRONNER (of Utrecht), *Centralbl. f. Nervenh. u. Psych.*,  
 April 1, 1905.

HEILBRONNER introduces the discussion of the above subject by the report of the case of an epileptic who, on several occasions, showed marked aphasic symptoms; these latter on one occasion followed a severe epileptic psychosis, on other occasions were merely preceded by ordinary epileptic attacks or a series of the same. This fact suggests that systematic examination of patients after such attacks would show such symptoms to be more common than is generally supposed.

The aphasic disorder was of the nature of an amnesic aphasia, and there was nothing in the general mental condition of the patient to account for it: the patient was accessible, showed good grasp, attention, and retention, had no special feeling of fatigue.

The aphasia consisted in the fact that patient was frequently

unable to find the name for various objects and pictures; the disorder was slight, for in spontaneous speech no poverty of his store of words was observed. As a rule, on the onset of an exacerbation the number of missing words was greatest, and gradually diminished; common objects caused least difficulty, objects were more easily named than pictures. This occasional difficulty in naming things was the only speech disorder, and was accompanied by no difficulty in understanding spoken language.

Such aphasic phenomena may occasionally be observed in more strictly organic brain lesions (softening, etc.), and Heilbronner has observed amnesic aphasia as an isolated brain symptom after concussion; it may also occur as the residual of a transitory sensory aphasia.

The diagnostic value of the symptom is discussed; the author calls attention to its occasional occurrence in general paralysis, where it presents essentially the same character as in epilepsy, and therefore is of no aid in separating the two conditions. Hysterical patients in the dream-state of Ganser may present the identical symptom, and the diagnosis must be made on the other symptoms of the two diseases.

In order to understand the nature of aphasic disorders in epileptics, one must also consider those cases of aphasia and asymbolia, following epileptic attacks or psychoses, which pass into well-marked dementia. The present case is a transition case between the above and cases of transitory impairment, without permanent defect. One must assume, on the analogy of general paralysis, that the aphasic disturbances of epileptics depend on a localised process, or at least upon a special increase of the morbid process in definite areas. Hitherto the nature, rather than the localisation, of the morbid process has been the object of investigation; the speech disorders suggest the locality of the most marked action of the specific process, and therefore such cases are particularly worthy of accurate pathological examination.

The motor cortex has been the usual object of examination. But convulsions, especially if general, are of little localising value; it is not the irritative phenomena, but the defect phenomena persisting for some time after the attack which should suggest the seat of damage. Unfortunately amnesic aphasia does not point to one definite area, and motor aphasia after epileptic attacks is rare and transitory; the seat of the lesion indicated by the amnesic aphasia is in the speech reflex-arc at some point before Broca's convolution. More accurate clinical studies of epileptic dementia from this point of view may be of great help in directing the lines of anatomical investigation.

C. MACFIE CAMPBELL.

**THE CLINICAL INVESTIGATION OF THE MUSCULAR SENSE.**

(330) (*Sur l'exploration du sens musculaire et la corrélation de ses diverses modalités.*) CLAPARÈDE, *XIV<sup>me</sup> Congrès des Médecines Aliénistes et Neurologistes*, Pau, August 1 to 7, 1904.

THE muscular sense is difficult to investigate for several reasons : (1) It is not a specific sense manifested by a single form of sensation, but comprises various conditions (modalités) which have to be examined in turn—the sense of weight, the sensation of movement, the stereognostic sense, etc. (2) We are still ignorant of the peripheral physiological factors of these diverse conditions—the dependence of the sense of weight, for instance, on muscular tension, articular pressure, traction on ligaments, and so on. (3) Cutaneous sensibility may, even when deep sensibility is absent, inform the subject of the position of the limb, or the weight of objects which he lifts. (4) The examination offers all the usual difficulties of hesitation on the part of the subject, contradictory results from successive observations, the influence of fatigue, etc.

M. Grasset has proposed to eliminate the effect of cutaneous sensibility by making the subject raise a weight by a thread which he holds between the finger and thumb. He is then asked to indicate whether he notices the lightening of the weight when this is supported by the observer. This method is faulty, because as soon as the weight is supported the subject's arm moves, and this movement is felt by the subject, even if the sensation of the lightening of the weight is absent.

The writer has, therefore, been experimenting with another method, which consists in placing two weights simultaneously, one in each hand, and noting any tendency to over or under estimation of the weight by one of the hands. For example, in over-estimation by the right hand, if two equal weights were used, that in the right hand would be reported the heavier.

Similar experiments were made with small wooden cubes of different sizes. One is placed in each hand and felt quickly by the subject, who then expresses his opinion as to their relative sizes.

No parallelism has been found between the sense of weight and the sense of volume; between the condition of cutaneous sensibility and the over or under estimation of weight or of volume; between the latter phenomenon and the motor power of the limb.

Further experimentation is requisite, but the method of examination made use of indicates very clearly and easily differences of sensibility between the hands which escape the usual methods of investigation.

W. B. DRUMMOND.

**THE IMPORTANCE IN CLINICAL DIAGNOSIS OF PARALYSIS (331) OF ASSOCIATED MOVEMENTS OF EYEBALLS (BLICK-LÄHMUNG), ESPECIALLY OF UPWARD AND DOWNWARD ASSOCIATED MOVEMENTS.** Presidential Address, Americ. Neurolog. Assoc. W. G. SPILLER, *Journ. of Nerv. and Ment. Dis.*, July and August 1905.

SPILLER has studied four cases with paralysis of lateral associated ocular movements, and nine cases with paralysis of upward or downward associated movements: he has examined microscopically the material obtained from four of these cases.

Hysteria may cause paralysis of associated ocular movements and there is no pathognomonic sign of this form of the palsy; the extreme difficulty of making a correct diagnosis is exemplified by a case shown before the Neurolog. Soc. of Paris on several occasions.

*Lateral* associated ocular movements. There is strong evidence that a centre for these is situated in the posterior part of the frontal lobe, but the evidence concerning a centre in the lower parietal region or angular gyrus is conflicting. Little, if any, clinical evidence can be found in support of Parinaud's opinion that, if the cortical centre for associated movements of the eyes in the frontal lobe be destroyed, the voluntary associated movements will be lost, but the reflex associated movements will be preserved. Hemianopsia cannot be regarded as the cause of conjugate deviation in all cases. Conjugate deviation of the eyes, due to cortical lesion, is always transitory. Persistent paralysis of lateral associated ocular movements indicates a lesion of the posterior longitudinal bundle near the sixth nucleus. In lesions of the pons, conjugate deviation of the eyeballs is not common, even as a late sign, when the lesion is confined to one side of the pons: it was absent in four cases examined by Spiller. According to Ransohoff, contracture of the internal rectus on the side of lesion indicates that the posterior longitudinal bundle of the opposite side is also involved; in four of Spiller's cases both posterior longitudinal bundles must have been much affected, and yet conjugate deviation did not occur. In four cases with autopsy the pupil was smaller on the same side as the lesion of the pons: this must be a sign of diagnostic significance, and it shows that the fibres controlling the iris pass through the pons. Nystagmus may be present on looking away from the side of the pons in which the lesion chiefly lies; this indicates pressure upon, or direct involvement of, the nucleus of the opposite sixth nerve.

The paper deals chiefly with paralysis of *upward and downward*

associated ocular movements. There is "little or no anatomical or pathological evidence that is really valuable" in favour of a cortical centre for these movements, but "in reasoning from analogy we must assume that such a centre or centres exist." "All the pathological evidence that I have been able to obtain in cases of persistent palsy of associated upward or downward movement is indicative of a lesion near the aqueduct of Sylvius." There is no proof of a centre for ocular movements in the corpora quadrigemina; clinical and experimental evidence is against it. There is no necessity to assume the existence of any supra-nuclear co-ordinating centre for ocular movements. For lateral movements, the posterior longitudinal bundles, and for upward and downward movements, certain fibres connecting different parts of the oculo-motor nucleus with one another and connecting the nucleus for the inferior rectus with the nucleus of the trochlear nerve, may be reasonably supposed to have co-ordinating function. In two cases of tumour of the pons there was impairment of upward associated movement, while downward movement was normal: the tumour did not invade the oculo-motor nuclei or implicate the corpora quadrigemina, but the posterior part of the oculo-motor nuclei was affected by pressure: this is evidence that the nuclei for the superior rectus and inferior oblique muscles are in the posterior part of the oculo-motor nucleus, behind the nucleus for the inferior rectus muscle. Isolated paralysis of downward associated movements (necessitating the implication of two separate nuclei) is strikingly rare compared with isolated paralysis of upward associated movement.

Full notes are given of Spiller's 9 original cases of paralysis of upward or downward associated movements, and abstracts of 38 cases recorded in literature: autopsy in 19 cases (tumour 15, bullet wound 1, apoplectic cyst 1, hæmorrhage 1, uncertain 1). Isolated paralysis of upward associated movement was present in 26 cases, and of downward movement in 5; both upward and downward movements were affected in 16 cases. In all 47 cases except one (Thomsen's), parts about the aqueduct of Sylvius were implicated. In Thomsen's case, in which there was paralysis of only upward movement, a gumma was found in the cerebral peduncles: the oculo-motor nuclei appeared to be normal, but Nissl's stain was not used. Until there is further proof, "we may well hesitate to believe that a peripheral lesion of one oculo-motor nerve may cause paralysis of only upward associated movements.

As regards prognosis, persistent paralysis of associated ocular movement of any form is usually a sign of grave significance, but one should be careful about giving a fatal prognosis. Seven of Spiller's forty-seven cases showed recovery, complete or partial—in some cases after the paralysis has lasted for years. The position

of the lesion, in the pons or about the aqueduct of Sylvius, makes these cases unsuitable for operation.

ASHLEY W. MACKINTOSH.

**A SIGN OF ORGANIC PARALYSIS OF THE INFERIOR EXTREMITY—ABILITY TO RAISE THE PARALYSED LIMB ALONE, BUT INABILITY TO RAISE SIMULTANEOUSLY THE TWO LOWER LIMBS.** (Un signe de paralysie organique du membre inférieur—Possibilité de soulever isolément le membre paralysé avec impossibilité de soulever simultanément les deux membres inférieurs.) J. GRASSET et A. GAUSSEL, *Rev. Neurolog.*, Sept. 15, 1905, p. 882.

IN certain cases of organic paralysis of the lower extremity, especially in hemiplegia, the authors have observed that when the patient is lying on his back, although he is able to raise the paralysed limb off the bed, he is unable to raise both lower limbs simultaneously. This point is interesting, since in cases of hemiplegia it is customary to find that movements carried out on the two sides of the body at the same time are usually better performed. The authors are of opinion that the inability to raise both limbs at the same time is due to defective fixation of the pelvis from paralysis of the pelvic muscles. They were able to prove this, for when the pelvis was fixed by pressing on the iliac spines it was found possible to raise both lower limbs from the bed simultaneously. The explanation is similar to that advanced by Babinski to account for the sign of "flexion of the thigh on the trunk" which he has described. The new sign which Grasset and Gausssel have observed may in certain cases prove of value in distinguishing between organic and functional hemiplegia.

EDWIN BRAMWELL.

**FURTHER CONTRIBUTION ON THE NATURE OF THE SO-CALLED SUPRA-ORBITAL REFLEXES.** (Weitere Beiträge zur Natur des sogen. Supra-orbital Reflexes.) CARL HUDOVERNIG, *Neur. Centralbl.*, Aug. 16, 1905, p. 740.

ON the grounds of its presence after successful removal of the Gasserian ganglion in four cases, which he reports, and from other observations which he does not mention in more detail, the author concludes that M'Carthy's "Supra-orbital reflex" is no true reflex, but is "dependent on the direct extension of the mechanical stimulation by periosteum, ligaments, and muscles, to the orbicularis palpebrarum," and suggests that the term "Eye-pheno-

menon" of Bechterew, or perhaps better, "Orbicularis phenomenon," would be more accurate and descriptive.

COLIN K. RUSSEL.

**EYE-STRAIN, ITS IMPORTANCE AND ITS LIMITATIONS.**

(334) G. L. WALTON. (A paper read before the Boston Society of Psychiat. and Neurol., May 18, 1905.)

WHILE adopting the position of the sceptic regarding the cure of epilepsy and chorea by glasses, the author is of opinion that "eye-strain" is one of the commonest exciting causes of headache in the otherwise healthy. It occurred to Dr Walton that some light might be thrown on the subject of headache and "eye-strain" by ascertaining the frequency of headache among those totally blind from infancy.

With this object in view he questioned regarding headache 100 individuals with healthy sight (of whom only 31 per cent. had never had headaches), 42 cases of partial blindness, acquired or congenital (of these 29 per cent. had never had headaches), and 90 individuals totally blind since infancy (66 per cent. had never suffered from headaches).

It would appear, then, that headaches are twice as common among those subject to eye-strain as among those in whom this element is wanting. In the opinion of the author, eye-strain is one of the most, if not the most, important exciting causes of migraine and migrainoid headache, and steps for its relief are imperative.

In no case has correction of refraction been given a thorough trial until (*a*) the glasses are properly centred, (*b*) their continued readjustment is practised, (*c*) the patient looks as much as possible through these centres instead of from side to side, (*d*) efforts are avoided at straining the eyes to see distant objects with the glasses, (*e*) spectacles instead of eyeglasses are used, and (*f*) the use of spectacles is constant, not intermittent. EDWIN BRAMWELL.

**CONTINUED ALCOHOLIC DELIRIUM OR CONTINUED HAL-**

(335) **LUCINATION IN CHRONIC ALCOHOLISM.** (*Déire alcoolique continu ou hallucinose continue dans l'alcoolisme chronique.*) SERGE SOUKANOFF, *Journ. de Neurol.*, July 20, 1905, p. 261.

THE author first briefly refers to the classification of various psychoses associated with the abuse of alcohol, mainly as to the precise ætiological relationship of these to alcoholism.

As one distinguishes melancholia and the melancholic state,



mania and the maniacal state, it is desirable to differentiate delirium tremens proper from a similar symptom complex which may be observed at the commencement of certain alcoholic psychoses, and also in the course of other mental affections where the alcoholic element is present. Thus Korsakoff's polyneuritic psychosis of alcoholic origin may begin symptoms like those of delirium tremens, as also may so-called alcoholic melancholia.

Delirium tremens may be seen in the course of chronic mental diseases, as in the initial phase of general paralysis, where the patient already affected by the disease abandons himself to alcoholic excess; or again, in the early period of dementia præcox. The syndrome, delirium tremens, may form the initial phase of continuous alcoholic delirium or continued alcoholic hallucination. In a former paper (in conjunction with Dr Wedensky) the author expressed the view that this disease should be placed in the category of autonomous alcoholic psychosis; that it should be distinguished from delirium tremens and from chronic alcoholic paranoia. From the latter it is to be differentiated by the preservation of the patient's personality, and by the circumstance that ideas of persecution are less marked or absent, while auditory hallucinations are very prominent. The affection is a continued but not a progressive one; and delusional ideas, while they do sometimes accompany it, have no tendency to evolution and systematisation. The patient generally maintains an attitude of criticism as to his auditory hallucinations. In view of its symptomatology, continued alcoholic hallucinations might be a more appropriate term than continued alcoholic delirium.

In illustration, an account of a case is given. The patient, a man of 34 years of age, with alcoholic family history, early learned to drink, and had indulged heavily before onset of his present illness. He was able to give a detailed description of his mental experiences at the beginning of the malady. The first symptoms were essentially those of delirium tremens—insomnia, varied hallucinations (including those of sight), general tremor, fear. At first the differential diagnosis of this as the initial stage of continued alcoholic delirium from true delirium tremens is almost impossible. In delirium tremens itself, auditory hallucinations may sometimes preponderate over visual, though their doing so ought always to raise a suspicion as to diagnosis. If the patient begins to complain of the voices being uniform, the same words and phrases being always repeated, one may look for continued hallucination rather than delirium tremens. As time goes on the diagnosis becomes simpler, the features suggestive of delirium tremens gradually falling into the background, while the auditory hallucinations alone persist, and that for years. It would appear that at least the great majority of cases are incurable. With time

the hallucinations become weaker and rarer. The patients retain their consciousness and personality, and are aware they are ill, can behave in an ordinary manner, and occupy themselves in their usual affairs. This disease should be distinguished from chronic alcoholic paranoia, the delirium in which develops towards systematisation. The latter is a progressive affection.

In the author's case of continued alcoholic hallucination, double purulent otitis media was present. While not denying that such a lesion may participate in the pathogenesis of this disease, it does not play the chief rôle. Marked cases may show no peripheral auditory lesion, and the treatment of such when present may not lead to improvement in the mental state. The abuse of alcohol may produce special functional trouble in cortical auditory centres.

In employing the term continued hallucination in chronic alcoholism, the author would indicate that it does not coincide with "acute Hallucinoze der Trinker" of Bonhoeffer and Wernicke, that corresponding rather to the "hallucinatorischer Wahnsinn" of Kraepelin, "Paranoïa alcoholistica" of Mendel, and "Paranoïa alcoolique subaiguë" of Serbsky. The classification of alcoholic psychoses into (1) acute alcoholic psychoses, and (2) states of dementia, recently suggested by Papadaky, does not appear to meet all the requirements of the case. By this method, alcoholic psychoses so special as delirium tremens and alcoholic dementia are placed along with an affection like Korsakoff's polyneuritic psychosis, which may have an ætiology other than alcohol. One must keep in view also that chronic auditory hallucination may be met with in peripheral lesions of auditory mechanism (Bechterew), and also in senile states where arterio-sclerosis is present, and the affection appears due to central causes. The relation of these cases of chronic auditory hallucination to the disease we have been considering requires further research.

A. HILL BUCHAN.

## PSYCHIATRY.

**ON THE RÔLE OF ATTENTION IN THE LOCALISATION AND  
(336) DEVELOPMENT OF HALLUCINATIONS.** (Ueber die  
Bedeutung der Aufmerksamkeit für Lokalisation und Ent-  
wicklung hallucinatorischer Bilder.) W. v. BECHTEREW,  
*Centralbl. f. Nervenh. u. Psych.*, May 1, 1905.

THE author discusses the question why a patient refers the imaginary voices heard to some definite external source, *e.g.* now to the floor, now to the wall, or to one organ rather than to another. External causes have a considerable influence in deter-

mining the point of reference of the voices heard ; in some cases voices are heard in any direction to which one directs the attention of the patient, even in the patient's own body. The author found in the case of an hysterical patient with suggested hallucinations, that the hallucinations were referred to any place towards which he directed the attention of the patient. The same result was reached both with auditory and visual hallucinations.

One can not only determine the localisation of the hallucinations, one can even call them up by increasing the attention in some cases, *e.g.* in alcoholics.

C. MACFIE CAMPBELL.

**THE MOTOR EXCITEMENT IN MIXED FORMS OF MANIC-  
(337) DEPRESSIVE INSANITY.** (*Die motorische Erregung im manisch-depressiven Mischzustand.*) K. PFERSDORFF, *Centralbl. f. Nervenh. u. Psych.*, March 1, 1905.

THE author reports the cases of three patients with depression and blocking of thought accompanied by certain motor excitement. While the excitement in such conditions is an independent phenomenon, the form which it takes depends to a certain extent upon the other symptoms ; it is this relationship which is analysed in the present communication. The movements, as a rule, were monotonous, aimless movements, frequently of the individual extremities ; although monotonous, they differed from the stereotyped movements of the catatonic, and were amenable to outside influences. The fact that the patients carried out on request complicated movements showed that the limitation of the motor excitement to the peculiar unrest was not due to the complete inhibition of complex motor ideas, and therefore could not be considered secondary to the thought difficulty.

As to the relation between the affective life and the excitement : where there was an almost complete absence of definite affect, play of facial expression was absent. In this condition grimacing appeared as a motor phenomenon, but this grimacing was never of the nature of facial expression ; when the neutral mood was replaced by a more variable emotional state, the grimacing disappeared, and was replaced by expressive movements. The unrest was most marked when there was least affect, and therefore could not be looked upon as secondary to the emotional condition ; in one case the monotonous restless movements of the period with neutral mood were replaced in periods of marked affect by coherent movements of expressive nature.

The analysis of such cases as are here presented is of importance

with regard to the question of the relationship of the anxiety-psychosis of the involution period to manic-depressive insanity.

C. MACFIE CAMPBELL.

### TREATMENT.

#### **THE TREATMENT OF CEREBRAL PALSIES AND ATHETOSIS (338) BY NERVE ANASTOMOSIS AND TRANSPLANTATION.**

W. G. SPILLER and C. H. FRAZIER, *Journ. Nerv. and Ment. Dis.*,  
May 1905, p. 310.

THE authors believe that there is probably a field for surgical intervention in certain selected cases of cerebral hemiplegia, but "whether any benefit will result to the hemiplegic individual or not, experience alone will decide," and they have no positive results to record. They think that, where it can be carried out, nerve anastomosis or transplantation is more rational than tendon-transplantation, as the former supplies a means of restoring the function of muscles previously paralysed. Surgery cannot help in cases of complete or nearly complete hemiplegia. But where, as is the rule, there is partial return of power in the paralysed limbs, and there is very unequal distribution of power in groups of muscles which oppose one another, the transference of a portion of nerve force from the stronger to the weaker group would seem a reasonable procedure. Thus, in the arm the flexors may alone or almost alone regain power, and their action is weakened by loss of extensor power; in such a case, the central ends of some of the least important of the flexor nerves might be anastomosed with the peripheral ends of extensor nerves, and the normal relation between the flexor and extensor muscles might be thereby more nearly restored. In one hemiplegic woman, with very slight return of extensor power compared with flexor power in the arm, the median and musculo-spiral nerves were exposed, and a flap, composed of one-half of the median nerve, with its base central-wards, was implanted into the musculo-spiral; no certain improvement can yet be recorded. Spiller has studied the conditions of paralysis in twenty-six cases of infantile hemiplegia, and finds that in eleven the flexors of the hand were so much stronger than the extensors that a nerve anastomosis might properly be attempted, but that only four cases offered much hope for improvement from an operation on the foot.

It is also suggested that, in athetosis, the power of the involuntary movements might be lessened by division of one or more of the motor nerves of the affected limb and immediate suture of the divided portions, or by leaving some of the nerves divided, or

—in cases where the flexors of the hand and fingers are unusually strong—by joining the peripheral end of a flexor nerve with the central end of an extensor nerve, and *vice versa*.

Dr Frazier also briefly indicates the lines and results of experiments on dogs in the way of "anastomosing the nerves presiding over one group of muscles with those of another."

ASHLEY W. MACKINTOSH.

**THYROIDECTOMY FOR EXOPHTHALMIC GOITRE.** FRANK (339) HARTLEY, *Ann. of Surg.*, Vol. xlii., 1905, p. 33.

HARTLEY records fifteen cases of exophthalmic goitre treated by thyroidectomy. The operation should not be reserved for the severer cases. The earlier the operation the better the condition of the patient to withstand the ordeal. The removal of one lobe and the isthmus is usually sufficient; but when the hypertrophy is vascular and bilateral, the symptoms are best relieved by removal of one lobe, the isthmus, and a part of the opposite lobe. In some instances the superior or the inferior thyroid artery of the opposite lobe was tied in addition to the unilateral thyroidectomy.

One patient died during the operation. The remaining fourteen patients, seen at intervals of time varying from one month to seven and a half years after operation, were in good health and able to work at their avocations without discomfort. They had all lost their anxiety and nervousness. The muscular tremor and, in all but three cases, the exophthalmos had disappeared. In no instance had the symptoms recurred. Thyroidectomy compares favourably with sympathectomy.

W. T. RITCHIE.

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## Reviews

**ON FLIGHT OF IDEAS, DEFINITION AND PSYCHOLOGICAL ANALYSIS.** (Ueber Ideenflucht. Begriffsbestimmung und psychologische Analyse.) H. LEIPMANN. Carl Marhold, Halle a. S., 1904, pp. 84, M. 2.50.

VARIOUS theories have been put forward as to the nature of the disorder which leads to flight of ideas. Liepmann clears the way for his own statement of the problem by a preliminary examination of these theories.

Ziehen sees the essence of flight in the increased rapidity of the association of ideas. But a discourse, if rational in itself, does

not become flighty, no matter how quickly it is produced, and experimentally it has been shown that the association-time of manics is not shortened.

For Aschaffenburg flight of ideas is secondary to loquacity, which is merely one example of the generally increased ease of psycho-motor discharge. But flight of ideas may occur without any loquacity, it may consist in a procession of mental images, and loquacity does not necessarily lead to flight, *e.g.*, in the catatonic. Kraepelin and Werincke both consider that the disorder is not psycho-motor, but intrapsychic; to determine its nature Liepmann undertakes the analysis of rational thought as opposed to flight of ideas.

He criticises Kraepelin's view that the difference between the two lies in the insufficiency of goal-ideas in flight of ideas. It is true that in some forms of reasoning we do strive to some definite goal consciously anticipated, which may be looked on as the connecting bond between the successive contents of thought; but this formula is quite inadequate, and does not apply to all forms of thought. Various attempts have been made to explain the various steps in connected thought as resulting from the "laws of association"; but thought is more than a mechanical association of ideas, and no matter how one strings together ideas according to these laws one cannot produce connected thought.

Liepmann sees the essence of connected thought in the fact that the various links in the chain of ideas are determined by the content of some dominant idea or theme, which may be either an idea of some actual phenomenon or an idea of more or less abstract relations. In expounding a subject in a logical manner it is the presence of this main theme, or of ideas which come under it, that gives connection to the train of thought; on the other hand, in flight of ideas the content of thought at any moment is determined either by a sense-impression or by the previous associations of the preceding idea.

The activity of the attention is the mechanism by means of which a theme or dominant idea gives successive contents of thought their connected stamp. The extent to which the dominant idea or theme keeps ideas related to it in the focus of consciousness at each moment is identical with the fixity of attention; attention to a certain content, and the dependence of that content upon a dominant idea, are merely two forms of expression for the same fact. Flight of ideas, depending upon the absence or inefficiency of such dominant ideas, is, therefore essentially a disorder of the attention. The tendency, common to the normal person and the maniac, to pass from one content of thought to other contents which have been previously associated with the first, is counterbalanced in the normal individual by attention

concentrating on those contents bearing upon the theme under consideration; while in the manic there is no such counteracting activity of the attention, or at least it is very much diminished owing to the subsidiary part played by dominant ideas. The attention of the maniac dwells for a very short time on any one content, is extremely fickle, distracted by casual associations and sense impressions, but when attracted by a new content it turns to it with great avidity: it is this combination which is characteristic of flight; where attention is at once feeble and fickle, as in certain demented cases, there is no flight of ideas.

Liepmann next passes to the apparent contradiction between the statements of patients that so many thoughts pass through their heads, and the results of Aschaffenburg, who found that the association time of manics with flight of ideas is not shortened. The explanation lies in the more superficial view of the manic; he attends to a content of thought not in its wider significance, but in its surface relations, and dwells a shorter time on each individual content; the time of passage is not shortened, but he makes shorter stops.

As to the word associations which play such a large rôle in manic utterances, Liepmann calls attention to the fact that the spoken word acts as a stimulus in two ways, through the muscular mechanism and the sense of hearing.

Occasionally manics run over a whole series of objects, such as the articles in a room, the varieties of a species: here, obviously, there is a dominating idea, viz., that of the species. The flighty character of such an enumeration is seen, according to Liepmann, in the fact that the theme or dominating idea is not itself related to any system of dominant ideas, in other words that the enumeration is not in any way a suitable reaction to the situation.

In this work Liepmann shows the value of psychological analysis in psychiatric work. The view that flight of ideas is due to a disorder of attention is a great advance towards a correct appreciation of this symptom. Although the author criticises severely the associationists, he tends himself to fall into their language, and his view of the mechanism of normal connected thought as consisting of a hierarchy of ideas is no more adequate than the view of Kraepelin, who sees the guiding principle in goal-ideas. Liepmann's view seems due to the fact that he takes a well-ordered exposition of a subject as the type of connected thought. To other forms of thought equally connected his scheme does not apply.

Of course one can always find some dominant idea which one can say is the connecting link in a connected train of thought. It is the connecting link to the person criticising the thought and seeking a logical justification of it, but was it present to the

thinker? It need not always be so, according to Liepmann himself, who talks of its influence from the sub-conscious sphere: its effect is seen in the attention paid to various contents of thought, eliminating the casual associations which tend to displace them. In other words, even in the absence of the dominant idea, the interest of the individual may be sufficient to keep the attention concentrated. When this is the case, it would be better to lay emphasis on the actual fact of interest rather than to give an artificial explanation by means of psychological abstractions. The primary fact is that certain contents of thought interest the individual more than others. His interests are the product of natural constitution and education; they are the active aspect of the personality; they represent thought as the reaction of an organism and not as mere impressions on a *tabula rasa*. When, as in the manic, there is a change in the personality, and these interests fade, there is no longer any check on the tendencies of which the laws of the association of ideas are the expression. Liepmann himself calls attention to the fact that certain remarks may be flighty and improper in the lecture-room which are absolutely proper in familiar conversation, because in the former they show a weakening of the dominant interests.

It is important that in the discussion of these problems one should avoid artificial modes of statement, for much confusion arises from regarding ideas as entities, and not as mere convenient abstractions from the concrete thought of an individual with a system of interests.

C. MACFIE CAMPBELL.

**DE L'HYPOCHONDRIE: ÉTUDE PATHOGÉNIQUE ET NOSOLOGIQUE. (Hypochondria: A Pathogenic and Nosological Study.)** P. ROY. Rennes: F. Simon. 1905.

THIS monograph was presented at the Congress of Alienists and Neurologists held at Rennes in August of this year. While not exhaustive, it gives a good critical digest of previous work, and summarises clearly the present opinion on the subject. The treatise is divided into two parts: the first, occupying all but a few pages, being devoted to a study of the pathogenesis of the hypochondriacal idea; the second dealing with its nosology. The first part is further sub-divided into various chapters. At the outset there is an exposition of the doctrines held up to the present day regarding the disease. These doctrines range themselves mainly under two adverse headings: Is hypochondria a neurosis or a psychosis? As types of these two doctrines, the author gives the views of Dubois (Berne) and of Head (London). The former believes that it is a functional disease, a psychosis, without any



brain lesion, not dependent on any organic peripheral disturbance, but owing its origin to a particular psychical constitution. Head, on the other hand, from an investigation of a number of specially instructive cases in whom insanity and other nervous diseases were absent, inclines to the belief that hypochondria and other allied mental manifestations are due to the reflected pain of visceral diseases. Normally visceral sensations are outside consciousness; but when reflected visceral pain makes its appearance, the abnormal sensations at once produce a change and usurp the chief place in the attention.

The author then passes to a critical review of the facts regarding hypochondria, which seem to him to be incontestably proved. He first turns attention to disturbances of *kainæsthesia* and emphasises their important relation to the emotional tone of the individual. Visceral disturbances next claim notice. In these cases there is a material basis for the hypochondriacal state. In this connection the importance of the brain constitution becomes more than ever manifest in all hypochondriacs. The pathological facts relating to the great sympathetic system are there mentioned. They are seen to be few, but of great importance. The next chapter is concerned with the cerebral troubles of hypochondria. This is universally recognised to be of the utmost importance in the pathogenesis of the condition, and by many is regarded as the sole necessary factor. The author, however, does not wholly agree with this, and in the following chapter states his view that both cerebral and visceral disturbances are necessary for the production of the disease. The treatment is then considered under the headings moral, medicinal, and surgical. The importance of the first is insisted on, the other two yielding only poor results.

The second part of the book, the final chapter, is devoted to the nosological study of hypochondria. The two necessary and sufficient conditions for the production of the disease are more or less decided disturbances of *kainæsthesia*, along with a mental habit tending more or less to the introspection of bodily sensations. In considering the differential diagnosis, attention is directed to the fact that many, if not the majority of, melancholic general paralytics present marked symptoms of hypochondria. This is to be accounted for by the frequent concurrence in them of both the somatic and psychical troubles which are specially necessary for the development of the hypochondriacal state. On the last page is given an admirably clear and concise summary of the author's conclusions. He denies the existence of hypochondria as a distinct and autonomous disease. "There are only symptomatic hypochondriacal states, which may be observed in the course of all those affections associated with a special psychical constitution disposed

to the interpretation of kainæsthetic disturbances. The different clinical manifestations of the hypochondriacal state may be explained by the predominance of the psychical element or of the kainæsthetic element in the diverse affections of which this state is symptomatic."

JAS. MIDDLEMASS.

**THE TREATMENT OF NERVOUS DISEASE.** By J. J. GRAHAM BROWN, M.D., F.R.C.P., F.R.S.E., Assistant-Physician to the Royal Infirmary. Edin. and Lond.: William Green & Sons. 1905. Price 15s.

THIS work is stated by the author to be an expansion of certain lectures delivered to senior students of medicine in the medical wards of the Royal Infirmary, and which appeared in an earlier form in the *Scottish Medical and Surgical Journal*. Although not actually delivered as lectures in this expanded condition, the lecture form has been preserved, which gives an added interest to the perusal of the book, as it has enabled the author to explain his views in greater detail than could be done in a more formal work. The idea underlying the arrangement of the lectures is that the physiological function of the part of the nervous system affected determines the special mode of treatment required, and that consequently the lesions of any set of neurones of identical or analogous function call for lines of treatment which have much in common. On this basis the various nervous diseases have been grouped as regards their treatment. While the author admits that it is not easy to carry this doctrine out with absolute consistency, he claims that it is a convenient method, enabling the whole subject to be more concisely treated than could otherwise be done. We agree with him, and congratulate him on having written such a suggestive and stimulating work, so well up to date alike in its statement of anatomical, physiological, and pathological facts, and embracing most of the modern forms of treatment of recognised value. One of the great merits of the work is the detail and care with which the various lines of treatment are described and the reasons for or against them explained. We cordially commend the work alike to the neurologist and to the general practitioner.

ALEXANDER BRUCE.

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\* A number of references to papers on Treatment are included in the Bibliography under the Individual Diseases.

# **Review**

of

## **Neurology and Psychiatry**

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### **Original Articles**

#### **A CASE OF LOCALISED DOUBLING OF THE SPINAL CORD.**

By ALEXANDER BRUCE, M.D., F.R.C.P.E., Physician to the Royal Infirmary ;

STUART M'DONALD, M.A., M.D., Pathologist to the Royal Infirmary ;  
and

J. H. HARVEY PIRIE, B.Sc., M.B., Ch.B., Clinical Tutor in the Royal Infirmary.

THIS remarkable lesion in the spinal cord was discovered accidentally during the course of a post-mortem examination on a patient who died of diabetic coma. The patient was a clerkess aged 31, unmarried, who had been under the treatment of one of us (A. B.) in the Royal Infirmary, Edinburgh, for six months for diabetes. The history given by the patient was that she had enjoyed good health all her life until about six months before admission to hospital, when she became weaker and began to suffer from polyuria, which was ascertained to be due to diabetes mellitus. During the period in which she was under treatment she was repeatedly and thoroughly examined as to the condition of her nervous system, mainly with the view of ascertaining the presence or absence of peripheral neuritis. It is therefore certain that there were no motor or sensory symptoms relating to the lower extremities. As the patient became weaker during the rapid course of her disease, her gait became gradually more and more feeble, but it presented no other abnormal characters. The deep reflexes in the lower extremities were also somewhat



feeble, but they were not abolished, and there was no abnormality of the plantar reflex. No sensory or vaso-motor disturbances were at any time detected in the lower extremities, or indeed any symptom that could be attributed to the abnormality of the spinal cord.

At the post-mortem examination, which was made by Dr Stuart M'Donald, the following conditions with regard to the spinal cord were noted (Fig. 1):—"Towards the lower end of the cord there is a remarkable malformation. In the lumbo-sacral region, the cord bifurcates into two distinct trunks, which lower down again unite into a single cord. The two trunks are entirely enclosed in a single dural sheath. Where the division is total, however, a fold of dura mater projected for a certain distance backwards between the halves, not reaching the posterior layer, however. At several points, especially at the upper part, some delicate filaments of connective tissue unite the pial coverings of the two halves. The first indication of the division is seen in a distinct flattening of the cord in the lower dorsal segments. In the same situation there is a distinct mesial groove on the anterior and posterior surfaces of the cord. This flattened condition is well marked in the first lumbar segment; the grooves rapidly deepen from this point downwards, and the division is complete less than an inch lower. The cord is completely divided into two parts for about two inches. At the lower end the cords unite just as they separated above. The extreme end of the cord and filum terminale appear normal. Above the bifurcation the grey matter appears to the naked eye to be arranged normally. In the right half the appearance on section is that of a complete cord; on the left side the grey matter is much more irregularly arranged. There was no abnormality of the bony canal either behind or in front of the cord.

"*The Size of the Halves.*—In the first lumbar segment the cord measures about 12 by 4 mm.; where the halves are again uniting is the maximum measurement—about 15 by 11 mm. The right half measures 10 by 6 mm., and the left half 9 by 6 mm."

The state of the spinal ganglia and of the anterior and posterior roots as they passed through the dura was unfortunately not ascertained.

The spinal cord was carefully hardened in 10 per cent.

formalin solution, and after being divided into small blocks these were imbedded in celloidin and cut into sections by means of a sliding microtome. Every tenth section of each division was stained by the Weigert-Pal method, and another similar series by Van Gieson's method.

The first departure from normality observed in the cord was about the lowest part of the eleventh dorsal segment, and consisted of a remarkable flattening of the cord so that the transverse diameter was more than twice the antero-posterior; of a transverse elongation of the central canal; and an excessive broadening of the posterior columns, which resulted in a great divergence of the posterior cornua.

In the first lumbar segment at its lower part, the central canal, still elongated, showed an irregular dilatation, with projections of connective tissue into its lumen. Where these projections were most marked the epithelium of the central canal tended to become lost, and it was obvious that the central canal was on the point of being divided so as to form several subsidiary channels. With the increase of the tissue round the central canal at a slightly lower level, the two extremities of the canal became cut off from the main and more central part (Fig. 2). The former produced small, fine canals, separated about equal distances from the middle line; the larger portion formed an almost tri-radiate opening surrounded by a thick mass of connective tissue, but before the cord became divided into two this primary central canal ceased to exist, ending blindly in a mass of ependymal cells a little below the point where the two lateral canals had branched off (Fig. 3). A fine band of connective tissue ran from each of the lateral central canals towards the anterior part of this larger mass. At the bottom of the antero-median fissure, lateral projections passed outwards on each side almost to the level of the small lateral central canals.

In the lower part of the first lumbar segment the two halves of the cord became separated from each other (Fig. 4). Between them there was a broadish band of somewhat loose connective tissue, in the middle of which lay a large cavity. At a first examination this was mistaken for a central canal which had become so dilated as to break through the anterior and the remains of the posterior commissure. Further observation showed, however, that the space was a dilated blood-vessel, and

not a central canal as had been supposed. At this level the cord appears at first sight to have been simply separated into two almost symmetrical halves, each of which contains an anterior and a posterior cornu with related white matter and central canal, the whole being surrounded by pia mater somewhat thickened. The pia mater projects laterally into the inner aspect of the cords for a considerable distance, almost as far down as the central canals on each side. Among the white nerve fibres on each side a narrow band of connective tissue passes from the position of each central canal inwards and somewhat backwards, and reaches the inner surface of each cord immediately behind the large vascular space referred to. These bands of connective tissue correspond in position to the similar bands described in the previous paragraph as ending in the mesial block of neuroglia, and at lower levels they will be replaced by grey matter of anterior and posterior cornua. As the sections are examined in a descending direction, the inner part of this connective tissue band becomes first changed on the left side of the second lumbar segment into a structure which corresponds in character to the gelatinous substance and posterior horn (Fig. 5). In front of this there appears also a small area of grey matter, which, as it contains polygonal cells resembling the ordinary motor cells in every respect except their slightly lesser size, must be regarded as a rudimentary anterior cornu. At slightly lower levels the two cords still remain separated, although irregular bundles of connective tissue pass between their pia mater sheaths. The right side (Fig. 6) in the upper part of the third lumbar segment presents a definite posterior cornu and a rudiment of an anterior cornu, while on the left side the grey matter representing posterior and anterior cornua has increased somewhat in size. It will be noted in this as in lower sections that the cornua on this inner side are more distinct on the right than on the left side. The anterior median fissure is now distinct on both sides, and they are almost on the same transverse plane. At this level it is quite evident that we have to deal not with a cord divided into two halves, but with two distinct cords. In these two cords the anterior and posterior cornua, which are situated on the outer sides, are practically normal in appearance, and they obviously constitute the downward continuation of the corresponding cornua of each

half of the cord in the dorsal region before the division took place. The grey matter on the inner side consists of rudimentary anterior and posterior cornua of new formation. The posterior cornua are recognisable as such by the naked-eye appearances of their gelatinous substance. The anterior cornu on the right side presents a certain resemblance to an anterior cornu, and it contains a small number of motor cells. That on the left side is quite irregular in form and its nature is only established by the discovery of a small number of motor cells. In the posterior columns there is as yet no posterior median septum.

As we trace the sections in a downward direction, we find that at the level of the fourth lumbar segment (Fig. 8) there are two perfectly recognisable cords, that on the right side as hitherto, and as will be seen in lower segments, being the more perfectly formed. In the figure the inclination of the two cords has been slightly altered in the process of mounting, and the angle of inclination of the two anterior cornua diminished. The left cord has been manifestly altered in shape by pressure. The gelatinous substance of the inner posterior cornu has developed into an irregular, almost tumour-like mass, and the anterior cornu has become considerably depressed and distorted and is only recognisable as such by the discovery in it of large motor cells. The anterior and posterior cornua on the right side, on the other hand, are perfectly recognisable, and a posterior septum can be traced from the side of the gelatinous substance towards the posterior surface.

In the first and second sacral segments there is an obvious attempt at re-union of the two cords. They lie closely together and are united by a much more considerable amount of highly vascular connective tissue. The anterior and posterior roots are, as formerly, seen in relation to the outer and more perfect halves of the two cords. Posterior roots can be distinctly seen entering into the posterior cornu in the inner half of the right cord, and the posterior columns are divided into two nearly equal halves by a distinct septum. The anterior cornu, although formed, is in a somewhat rudimentary, but still recognisable condition. In Fig. 9 a projection in which lies anterior cornu tissue is seen on the inner half of the left cord. This is the beginning of the re-uniting bridge between the two cords. The inner posterior cornu on the left side is large in the first sacral segment, and

has swollen into an immense tumour-like mass with peculiar lateral projections in the second sacral segment (Fig. 11); the anterior cornu here being no longer recognisable in the section.

In Fig. 10, which corresponds to the lower part of the second sacral segment, the irregularity of the two cords is well shown. Seven central canals are present, one distinctly seen on the right side being a large cavity with thickened neuroglial tissue round it, a second one forming an irregular mass in the projecting tongue-like process of the right cord. Within the left cord there are five central canals or collections of ependyma cells placed behind each other. By the time the third sacral segment is reached, the two cords have reunited. The union has taken place in a somewhat peculiar manner. A first glance at the section suggests that the anterior cornu of the left half has been peculiarly distorted, but that otherwise there is only a single cord present. A more careful examination shows, however, that this is not the exact state of affairs, but that there are still real portions of the two cords conjoined. Two crosses (x x) have been placed on Fig. 12, and if a line be drawn between these, all that lies to the right of that line corresponds to the right cord, all that lies to the left corresponds to the fused part of the left cord; the two posterior cornua, distinctly recognisable by their gelatinous substance, belonging to the right cord. In front of the left gelatinous substance an area of grey matter represents the remains of the left anterior cornu, driven somewhat out of place by the pressure of the other cord. Large motor cells were found along the posterior border of this grey mass, putting its nature therefore beyond doubt. In the part of the cord found to the left of the line joining x to x, nerve cells were found in the anterior part of the grey matter, and also a group in the small projection immediately to the left of the oblique bundle of fibres running in the line between x to x. This group seemed to be the post-postero-lateral group, and the fibres those of posterior nerve roots, but there was no trace of a posterior cornu. A small collection of ependymal cells underlay the short diverticulum of the anterior fissure which comes off just in front of the anterior x. A large and distinct central canal was found in the right half of the cord. At the extreme lower end of the cord, as indicated by the last figure, the same relation of fusion of the two cords still persists, and it was found that

two central canals could be traced to the extreme end of the filum terminale.

With regard to the explanation of this case, there could be no manner of doubt whatever that this was not a case of artefact, such as has been shown by Van Gieson in the *New York Medical Journal*, Vol. lvi., 1892, 'to be so often the cause of alleged malformations. It was equally certain that the cord was not merely split into its two halves, as appeared to us to be possibly the case on first examining the sections at the upper part of the division. There could be no doubt that there were two more or less perfect cords. A somewhat similar case has been described by Theodor (*Arch. f. Kinderheilk.*, Bd. 24, 1898); his figures show a remarkable similarity to those obtained in this case, with this difference, that the formation of the two cords has taken place before their separation from each other has become complete. Theodor gives references to a thesis by Steiner ("Ueber Verdoppelung des Rückenmarks," *Inaug. Dissert.*, Königsberg, 1895), but at present this is not available to us. Steiner has collected 35 cases of fissure of the spinal cord. To these Monakow in the *Verhandlung der Naturforscherversammlung*, 1896, has added another. These with Theodor's and ours make 39 in all. According to Steiner the cases may be collected into two groups, in one of which there is a greater or less splitting off of the spinal cord substance, and in the other a complete duplication of the cord or part of it. In this second group there are several cases by von Rechlinghausen, P. Foa, Bonome, Bernard, Feist, Chiari, Sulzer, Monakow, Steiner, and others.

As regards the manner in which the duplication has been produced, it is probably not of the nature of a teratoma from the inclusion of a portion of a second foetus, but the product of some change in an early stage of the development of the medullary plate. Some process must have taken place by which the medullary plate has been pushed forward by an upgrowth of tissue from below, so that two neural grooves and then two neural tubes have been formed. There is evidence from Hertwig's paper ("Urmund und Spina bifida," *Arch. f. mikros. Anat.*, Bd. 39, 1892) that this condition can be experimentally produced. He has found it possible to produce two neural

grooves, sometimes with one notochord, sometimes with two, in the ova of frogs. The upgrowth of the projection of the anterior fold of dura mater between the two cords would seem to indicate that the separation was dependent upon the excessive development of the mesoblast tissue in front of the neural plate.

The remarkable features of this case were the completeness of the double cord formation, particularly in one of the halves, the absence of any malformation of surrounding structure, and of any interference with the functions of the cord. It would have been very interesting to have followed out the course of the nerve roots, but unfortunately the cord was removed in its dural sheath from the vertebral canal before there was any suspicion of its malformation.

NOTE.—Whilst this paper is going to press, the writers have discovered another case of partial doubling of the spinal cord, which they hope to publish in an early number of the Review.

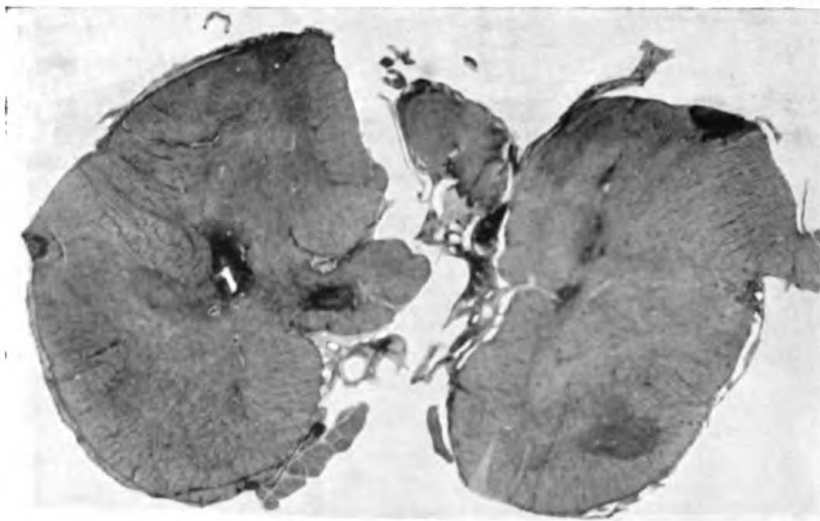
#### DESCRIPTION OF PLATES.

- FIG. 1 ( $\times 1$ ).—Anterior aspect of the lumbo-sacral portion of the cord showing splitting and duplication. A tag of dura mater can be seen extending through between the two cords.
- FIG. 2 ( $\times 5$ ).—Section at level of first lumbar segment, L 1: Van Gieson stain. Dilated central canal with projection into the lumen of the thickened neuroglial capsule and atrophy of the epithelium over these projections. On the right side a separate central canal has been cut off just behind the tip of the lateral prolongation of the anterior fissure.
- FIG. 3 ( $\times 5$ ) L 1.—Weigert-Pal stain. Two halves of cord almost separated. The anterior median fissure with lateral prolongations filled with pial sheath and intervening highly vascular fibrous tissue. Posteriorly the thickened pial septum is united to a block of neuroglial tissue reaching to the anterior commissure.
- FIG. 4 ( $\times 5$ ) L 1.—Van Gieson stain. Complete separation of the two halves of the cord. Large central vascular space. Left cord shows a prominent band of fibres stretching inwards and backwards from its central canal; on the right side this is also present, but feebly represented, appearing as a mere streak.
- FIG. 5 ( $\times 5$ ).—Probably L 2 upper. Weigert-Pal stain. Two halves completely separate. Between them the two pial sheaths with intervening vascular connective tissue: lateral processes dip into a fissure on either side, the anterior median fissure of the cords. In the left cord on its

FIG. 1.



FIG. 11.





1

2

PLATE 27.

FIG. 2.

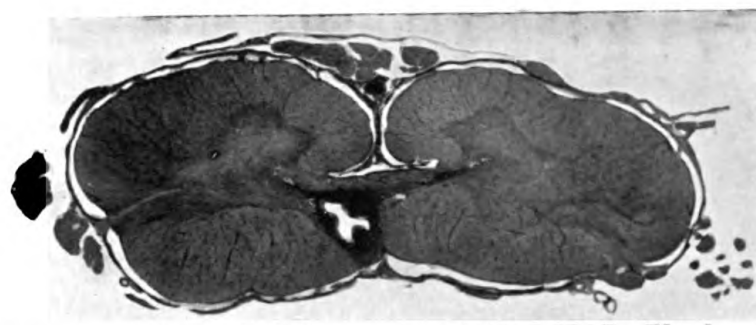


FIG. 3.

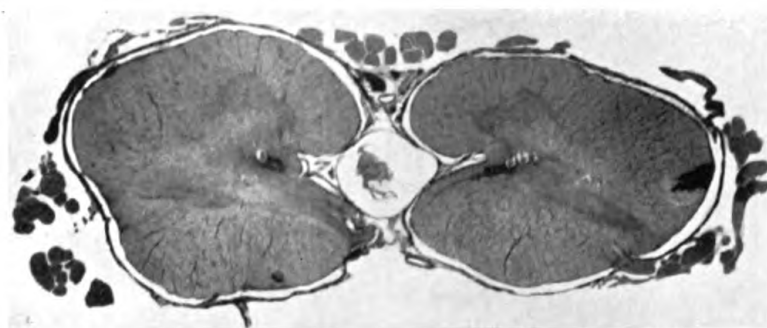


FIG. 4

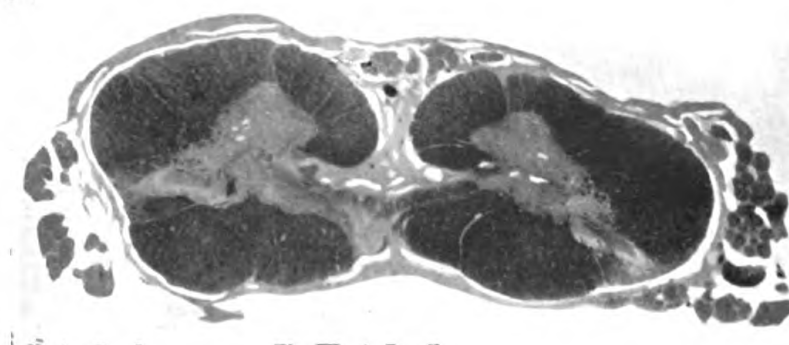


FIG. 5.

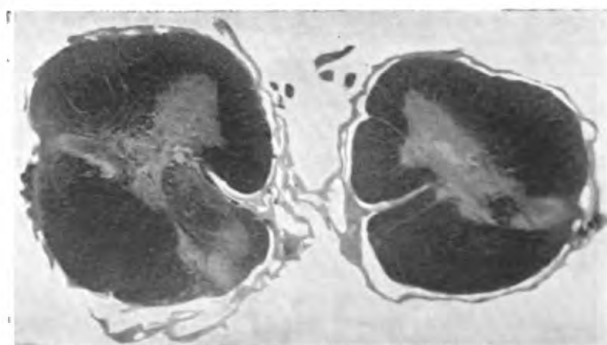




PLATE 28.

FIG. 6.

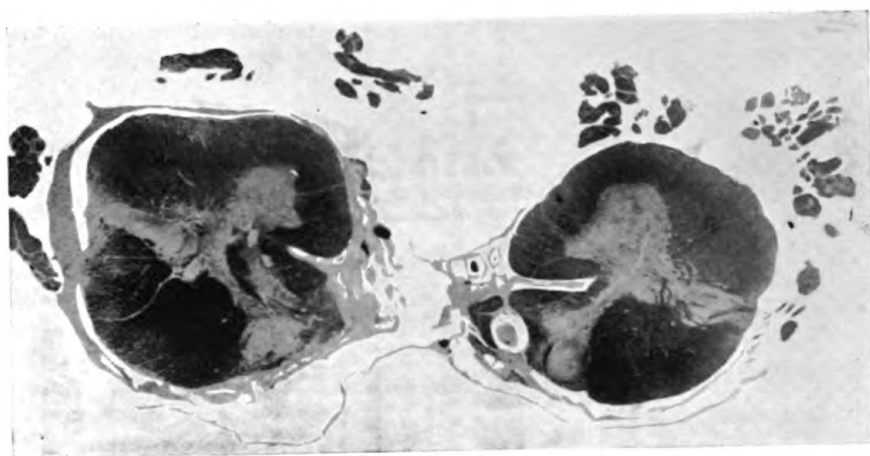


FIG. 7.



FIG. 8.





FIG. 9.

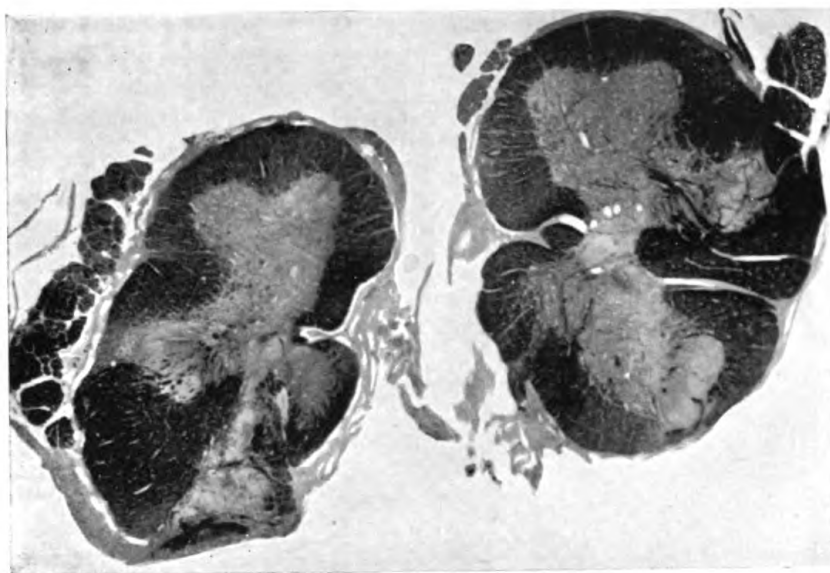


FIG. 10.



FIG. 12.

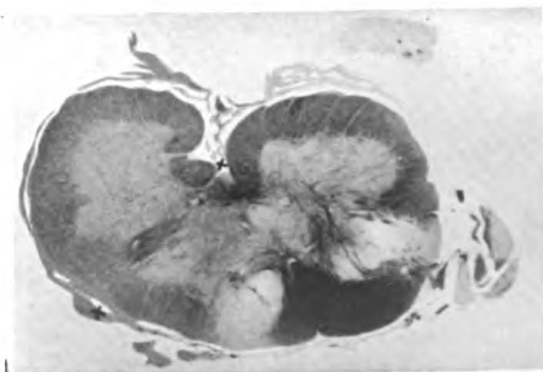
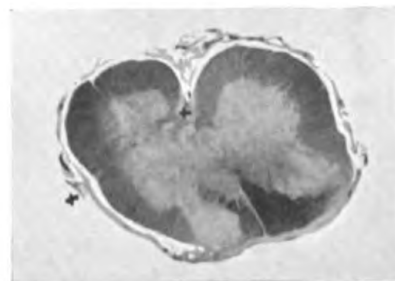


FIG. 13.





inner and posterior aspect a definite posterior cornu has appeared, indicated by the convoluted area of gelatinous substance. In front of this an area containing nerve fibres and strands of neuroglial tissue. In the right cord a delicate strand of neuroglia runs from the grey matter inwards parallel to the anterior median fissure.

FIG. 6 ( $\times 5$ ) L 3.—Weigert-Pal stain. The right cord shows the perfect outer half; the inner half has a fully formed posterior cornu, less perfect anterior cornu, and there is a separated island of anterior column. In the left cord the inner half shows compressed and crushed grey matter containing both anterior and posterior cornua, of which the latter only is distinctly recognisable. Well medullated anterior and posterior roots on both sides in relation to the outer halves of each cord.

FIG. 7 ( $\times 5$ ) L 3.—Weigert-Pal stain. Outer halves of both cords again appear normal. In the right cord the inner half now shows definite anterior and posterior cornua; the anterior is small, but it contained definite motor cells. A posterior septum divides the posterior columns into two irregular halves. The left cord shows branching anterior fissure; no definite posterior median septum; posterior and anterior cornua both present, altogether irregular in outline, only a trace of antero-lateral columns.

*Note.*—Figs 5 and 6 show the natural inclination of the two cords to each other. Into the angle between them a V-shaped fold of dura projected. In Fig. 7 the two outer and anterior halves have accidentally approached each other too much.

FIG. 8 ( $\times 5$ ) L 3.—Lower part near junction with L 4. Weigert-Pal stain. In the right cord the inner half is more nearly the size of the outer; the posterior septum is more nearly mesial in position. Distinct posterior nerve roots can be seen entering the posterior cornu of the inner half. The left cord has the inner half more deformed than in previous figures. Distinct afferent fibres seen in the posterior cornu.

FIG. 9 ( $\times 5$ ) L 5.—Weigert-Pal stain. In both cords the outer halves appear normal, although the left has been somewhat flattened obliquely. In the right half the anterior and posterior cornua of the inner side are distinctly recognisable; a few posterior roots pass inwards through the gelatinous substance, and some anterior roots pass outwards. The central canal is dilated. In the left half the posterior cornu is distinctly recognisable through its narrow neck and gelatinous substance; a separate anterior cornu with indefinite outline and separated by a layer of white fibres from the periphery.

FIG. 10 ( $\times 5$ ) S 2.—Van Gieson stain. Shows multiplication of the central canal. The main canal of the right cord is dilated and surrounded by a thickened neuroglia capsule. In the projecting lobule, which is the commencement of a bridge uniting the two cords, is another canal which branched off the main central one. In the left cord, in addition to the main canal opposite the bottom of the shallow anterior median fissure,



there are in the inner (irregular) half four other canals or collections of endyma cells, branches from the main tube.

FIG. 11 ( $\times 5$ ) S 2.—Weigert-Pal stain. On the right side an almost perfect cord, only the anterior cornu of the inner side being still somewhat defective in size. In the left cord the outer half is practically normal. The inner half is represented by an irregular tumour-like formation of grey matter, consisting entirely of posterior cornual tissue and gelatinous substance.

FIG. 12 ( $\times 5$ ) S 3.—Weigert-Pal stain. Below the junction of the two cords. The part to the right of the line between the two crosses (x x) represents the right cord of higher levels, and is a perfect complete cord, but with the left anterior cornu compressed and forced backwards by the part to the left of the crosses, which represents the left cord and consists of anterior and lateral white columns, a separate central canal at the bottom of the small anterior fissure, anterior cornu with several groups of motor cells, and posterior nerve roots entering, but no apparent posterior cornu.

FIG. 13 ( $\times 5$ ) S 4.—Weigert-Pal stain. Similar to Fig. 12.

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### A CASE OF TRANSVERSE MYELITIS IN A BOY OF FOUR YEARS OF AGE.

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MYELITIS is a rare disease in children. Under the age of ten years it is very rare. No doubt we must except from this statement cases due to compression of the cord, and cases of acute anterior poliomyelitis. Putting these cases aside, we find that myelitis is usually the result of injury, or appears as a complication in the course of some acute infectious disease.

The case about to be narrated is interesting from several points of view. In the first place the disease appeared at a very early age, the child having just entered his fifth year. In the next place, none of the ordinary causes were present. Indeed, we are unable to suggest any cause for the attack, though perhaps the diarrhoea from which the child suffered several times before the onset of his illness may be held to favour the theory of a toxic origin, the poison being absorbed from the alimentary canal. The apathetic mental condition which was noticed periodically might also be held as supporting such an explanation. In the last place, the very marked degree of im-

provement which took place under simple rest in bed is noteworthy, and would have delighted the distinguished author of "Rest and Pain." No form of apparatus was made use of to secure rest. The boy simply lay in bed. But a bed, as all old students of Professor Chiene's know well, is a splint. A bed is a splint, and for a child whose limbs were completely paralysed, no other splint was necessary to secure the complete rest which constituted the treatment.

The patient is the fourth child in a family of five, all strong, healthy children. The father and mother are healthy. No history of tubercle, syphilis, rheumatism, or nervous disease was obtained.

The boy himself seems to have been quite strong and well till about August 1904, when he had an attack of diarrhoea. He had another attack in October, and was attended by Dr Somerville of Galashiels, who found that there was some fulness of the abdomen, and the child was dull and apathetic. After a while he recovered, and was up and about again. The diarrhoea, however, recurred at intervals during the winter.

In April he was confined to bed by pain and swelling in one ankle. A few days later the other ankle became similarly affected. The nature of this pain and swelling was not very clear. It lasted for about three weeks, and during that time the child suffered from some itching (formication?) of the legs.

After the subsidence of the pain and swelling it was found that the legs were getting weak, so that the child could stand only with assistance. In a short time he lost power in his legs completely. At this time, also, he suffered from some difficulty with his bladder and bowels. When he tried to make water it was some time before it would come, and when it came it sometimes only dropped. The bowels moved every day, but he did not seem to have control over them. Nothing abnormal was noticed in the appearance of the urine or of the motions.

As his condition was getting worse, Dr Somerville sent him into the Children's Hospital, to which he was admitted on the 16th of June 1905.

His appearance at the time of admission is fairly well shown in the plate. He is a healthy-looking, well-nourished boy. As he sits upon the stool he has, owing to the weakness of his lower limbs and lumbar muscles, to support himself upon his

arms. His legs were not of quite such herculean proportions as the photograph represents, but there was certainly no wasting. A band of scratch marks around the trunk is quite evident in the illustration, and, doubtless, indicates a zone of cutaneous irritability of some description.

Upon examination at the time of admission the following points were made out:—

*General.*—Nutrition and colour are good. The alimentary, circulatory, and respiratory systems are normal. The urine also is normal. There is no swelling of any of the joints, and no œdema of the skin.

The boy lies on his back with his legs apart. The legs are very rigid, and marked priapism is present.

*Mental Condition.*—Although the child is dull and apathetic, and disinclined to talk or play, he is perfectly intelligent, and understands quite well what is said to him.

*Motor Functions.*—There is complete paralysis below the level of the umbilicus. The legs are very rigid. The knee-jerks are not easily obtained owing to the great rigidity of the limbs, but there is very marked ankle-clonus on both sides, and the Achilles-jerks are much exaggerated. The nutrition of the muscles is good.

The movements and reflexes of the upper limbs are normal. There is no alteration in the face nor in the eye movements, and the pupils react normally.

The electrical reactions of the paralysed muscles are normal.

*Sensory Functions.*—There is no evident anæsthesia. Round the upper part of the abdomen there is a broad zone of scratch marks, apparently indicating an area of some kind of cutaneous irritability (? hyperæsthesia).

*Organic Reflexes.*—The urine and fæces are usually passed in bed, but at times the child seems to be uneasy or pained during micturition, and may take a long time to pass a small quantity of water. Thus, on one occasion, he took twenty minutes to pass three ounces. Priapism is frequently present, and there is a tendency to handle the external genitals. The bowels are usually constipated.

*Spine.*—There is no deformity or rigidity of the spine, nor can any local tenderness be made out. In short, there is no evidence of spinal caries.

PLATE 30.





*Treatment and Progress.*—The treatment adopted consisted simply of rest in bed and suitable nursing. For a month after admission the patient remained practically *in statu quo*. Then he began to improve, and the improvement proceeded fairly rapidly. By the 6th of August he could move his legs, and could even stand for a few seconds without support. He had better control of his bladder and bowels, and usually avoided incontinence. By August 13 he could walk without assistance, and was also much more lively and playful. He was, however, still kept in bed.

Improvement continued steadily, but about the middle of September he seemed out of sorts for a few days. His temperature became a little irregular, and he was very dull and quiet, but made no complaint of pain. Nothing could be discovered to explain the fever, and it soon passed off. Improvement continued steadily.

By the end of September he could run about quite well, and was allowed to be up in the afternoon. The child was now looking very well, and the trouble with the bladder and bowels had completely disappeared. The knee-jerks, however, were still exaggerated, and ankle-clonus easily elicited. Babinski's sign was sometimes present, and sometimes not. This was the condition when the child was sent home in the beginning of October.

The diagnosis of transverse myelitis in the dorsal region of the cord in this case seems to be quite clear, but can we exclude a compression myelitis due to caries of the spine? There was no spinal deformity, but compression of the cord in caries may arise before deformity appears. In this boy, however, there was not only no deformity, there was no rigidity of the spine, and there was no tenderness. Moreover, we have no history of the acute radiating pains which usually occur early in caries from irritation of the nerve roots. The mode of onset of the illness, also, favours a diagnosis of acute myelitis, the paralysis having appeared during a febrile attack, concerning the nature of which the family doctor could not make up his mind.

In conclusion, I desire to thank Dr Somerville for notes of the child's illness before admission, and Dr Melville Dunlop, in whose ward the patient was treated, for permission to publish the case.

**DIPHThERITIC HEMIPLEGIA: A CASE WITH COMMENTARY.**

By J. D. ROLLESTON, M.A., M.D. Oxon.,

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A BOY, aged 6, was admitted to hospital on November 21, 1904, for diphtheria. He had been ill six days. The extensive faucial œdema and exudation, cervical adenopathy, rhinorrhœa, and oral fœtor testified to the severity of the attack. There was a cloud of albumin in the urine, which persisted till the forty-first day of the disease. On admission and on the two following days he received three doses of antitoxin of 24,000 units each. The throat was syringed four-hourly with a lotion containing potassium chlorate and myrrh, and he was given 5 minims of adrenalin solution (Parke Davis) in a drachm of aqua camphoræ two-hourly. On the 25th the voice was first found to be slightly nasal. The membrane had all gone from the fauces by the 26th, leaving extensive superficial necrosis of the mucosa of the tonsils, uvula, and soft palate.

The same day, on examination of the heart, which had hitherto been normal, the sounds were found to be very rapid and close together. The liver, which had been examined daily and hitherto shown no signs of enlargement, could now be felt one finger's breadth below the ribs. The voice was more decidedly nasal. On November 27, the pulses were weak and unequal. The apex beat of the heart was close to the nipple line, and there was triple rhythm. The liver was now one and a half fingers' breadth below the ribs. On November 28, general urticaria developed, fresh crops appearing during the next two days. The liver was two fingers' breadth below the ribs. The heart and pulses were as before.

On December 29, the fourteenth day of the disease, the patient vomited for the first time. The heart action was rapid. Triple rhythm with occasional irregularity was present. The apex beat was still in the nipple line.

The liver was now three fingers' breadth below the ribs. Mouth feeds and medicine were stopped, and nutrients containing 4 ounces of peptonised milk and 15 minims of adrenalin solution

were administered every four hours. The vomiting persisted, the pulses became weaker, the heart action more irregular, and the total amount of urine in the twenty-four hours fell to  $3\frac{1}{2}$  ounces.

On December 3, the eighteenth day of the disease, 20 minims of tincture of belladonna and 20 grains of potassium bromide were substituted in alternate feeds for the adrenalin solution, which was omitted.

The vomiting ceased, the enlargement of the liver began to subside, and the amount of urine increased, but the physical signs of the heart showed that that organ was considerably affected. As the vomiting had ceased, mouth feeds were resumed, consisting of whey, and bread and butter, and no further vomiting occurred.

On December 7, nothing was noted except that the child was very listless. The cardiac apex beat was in the nipple line, the action was very irregular, the sounds being of the nature of what Comby graphically calls *faux pas de cœur*. The enlargement of the liver had quite subsided.

On the 8th, the twenty-third day of the disease, the peculiar appearance of the child and his inability to use his right arm made the nurse send for me. On arrival I found well-marked palsy of the right lower facial muscles and deviation of the tongue to the paralysed side. There was complete loss of power in the right upper limb. In the lower extremity the palsy was less complete. Motor aphasia existed.

The knee-jerks were absent on both sides. Babinski's sign was present on the right side, the normal flexor response on the left. Ankle-clonus, which was first sought for the following day, was present and could be elicited till December 17.

The epigastric, abdominal, and cremasteric reflexes were deficient on the right, normal on the left side. Sensation did not appear to be affected. The urine as hitherto contained a cloud of albumin, but no sugar.

On December 9, the right foot and lower part of the leg were glossy and œdematous and the paralysis in the lower limb was more marked. Spontaneous movements were not performed, but the leg was drawn up when the sole was tickled. On the 10th, the twenty-fifth day of the disease, paralysis of accommodation was observed. The heart was now normal. On the 14th, very



slight spontaneous movement of the right foot was first noted. The upper limb was still in a state of flaccid palsy, and the aphasia persisted.

The improvement in the motor condition of the lower limb continued, until on December 28 he could move the right limb almost as well as the left. The great toe of the right foot was then found to give a flexor response, but not so well as on the left side.

On December 23, the thirty-eighth day of the disease, the child first began to cough over drinks, but no difficulty in swallowing solids was noticed till the 29th, when salivation, pharyngeal gurgling, and choking indicated paralysis of the muscles of deglutition.

Mouth feeding was therefore discontinued, and he was given 4 ounces of peptonised milk with 15 minims of belladonna by a nasal tube every four hours. The cot was raised to allow the excess of mucus and saliva which he could not swallow to drain through his nose. On January 3, twenty-six days from the onset of the hemiplegia, slight spontaneous movement of the right arm at the shoulder was first noted, and from that date gradual improvement in its movements occurred, though independent movement of the fingers did not take place till January 20, by which time he had so far recovered his power of speech as to be able to say and sing several nursery rhymes. On January 7, the clearer sound of the voice and the absence of salivation from the mouth, and of escape of mucus and saliva through the nose, indicated that the pharyngeal palsy was passing off. Soft solids were at first given by the mouth, and by the 9th he was able to take ordinary food well.

On January 11, the 57th day of disease, when he was first propped up in bed, the weakness of the neck muscles was shown by the head rolling from side to side or falling backwards. Massage of the back and of the affected limbs was employed and subsequently the faradic current as well.

Facial paresis lasted till within a few days of his discharge. Deviation of the tongue to the right continued till February 28. On January 21, the first day he was allowed up in clothes, he could walk with support, but it was not till February 1, the seventy-eighth day of the disease, that he could stand or walk by himself, even for a few steps. His gait at first in no way

suggested previous unilateral loss of power, but was that characteristic of pronounced diphtheritic pseudo-tabes. As the ataxy passed away, some dragging of the right leg was noticeable, which continued, though with gradual improvement, till the time of his discharge. Mental impairment, to the existence of which in diphtheritic hemiplegia Baginsky has drawn special attention, betrayed itself by an unusually vacant expression, which persisted after the facial palsy had disappeared, by torpor and apathy, and especially by amnesia. For some time after he had recovered his power of speech he would frequently forget his own name and address, the names of his nurses, and the letters of the alphabet. On one occasion feces and urine were passed in bed. A vigorous moral tonic was exhibited and incontinence did not recur. Improvement gradually occurred, but it was not until a month after he was first allowed up that he showed any inclination to take an active part in the games of the other children. When he left the hospital on March 23, 1905, after a stay of 123 days, his condition was as follows:—He could walk and run fairly well, though with a very perceptible limp. The fingers of the right hand could be moved at will, but were not able to grasp any object. There was no wasting of muscles nor contractures. The cutaneous reflexes were active on both sides, having already returned to the normal on February 14, the ninety-first day of the disease. The knee- and ankle-jerks, which had been entirely absent till the ninetieth day of disease, were only present on the right side. His speech was unaffected, and there was no obvious mental deficiency. Since leaving the hospital there has been slight improvement, but complete recovery has not been attained.

Though several cases doubtless pass unrecorded, hemiplegia following diphtheria is so uncommon that Slawyk (1), writing in 1898, had been able to collect only 50 cases. A search through literature has enabled me to add 15, thus bringing up the total to 65, including the present one, and another that has occurred subsequently at the same hospital. The earliest of these 15 cases is that of Sir W. Gull (2). Right hemiplegia developed, and was attributed by Gull to cerebral thrombosis. The hemiplegia remained for several months, after which there was slow improvement. Humphry's (3) case, a boy of 11, developed right hemiplegia about the fourteenth day of disease,

associated with paralysis of the palate, pharynx, and larynx. Death occurred from paralysis of the diaphragm. Post-mortem, a small area of suppuration with softening of the adjacent brain substance was found in the left cerebral hemisphere. Rosenthal's (4) case, a girl of 15, developed right hemiplegia in the third week after recovery from the initial angina. Paralysis of the palate and diplopia occurred at the same time. The skin on the affected side was insensible to electrical, thermal, and mechanical stimuli. In Hunter's (5) case, left hemiplegia came on in the third week, and was associated with cycloplegia, paralysis of the soft palate, muscles of the neck and back, and both legs. Sensation was normal. The superficial reflexes were diminished on the left side of the body, and both knee-jerks were entirely absent. Rigidity and slight atrophy of the left arm ensued. Recovery and some improvement occurred. The hemiplegia was regarded by Hunter as due to hæmorrhage into the right internal capsule. Wohlgemuth's (6) case was that of a girl aged 8. Right hemiplegia occurred at the thirty-second day. No cardiac disease was present, but much albuminuria. Recovery took place. Woollacott (7) says that in 4000 consecutive cases of diphtheria at the Eastern Hospital, hemiplegia occurred only twice. Both cases recovered. It is interesting to note in this connection that the present case is the first of the two solitary instances of hemiplegia that have been met with among 4407 consecutive cases of diphtheria that have been admitted to the Grove Hospital since it was opened in 1899. The other case was that of a girl aged 5. The initial faucial attack was very severe. Palatal paresis occurred on the tenth day. On the fifteenth the heart showed signs of dilatation. On the seventeenth, right hemiplegia occurred. On the eighteenth, death took place, ushered in by coma. Albuminuria was present on the seventh, and persisted till death. No autopsy was held, but cerebral embolism was probably the cause of the hemiplegia. In Mariottini's (8) case, a few days after the subsidence of the angina, endocarditis and left hemiplegia developed. Post-mortem, a focus of softening was found in the external capsule, corpus striatum, and internal capsule. The hemiplegia was caused in this case by an embolus due to endocarditis. In Breton's (9) case the girl, aged 3, had right hemiplegia and livid spots, followed by gangrene, on the left leg. The hemiplegia persisted for eight

months, though the child could walk by dragging her leg. There was doubtless in this case endocarditis and multiple emboli. Teillais (10) recorded 3 cases in children aged 6, 7, and 9 respectively, in which hemiplegia was associated with ocular and palatal palsies, and attributed the phenomena to cerebral hæmorrhage. Two recovered, one died. Knoepfelmacher (11) recorded a case of right hemiplegia, preceded by early affection of the palate, in a girl aged 4. Recovery took place, but the paralysis persisted. He regarded an encephalitis, with lesser probability an apoplexy, as responsible for the condition.

Of the 65 cases, 20 were males, 30 females. In 15 the sex was not given. The ages of the patients ranged from  $1\frac{1}{2}$  to 15 years. Right hemiplegia occurred in 38, and left in 22. In 5 no details were given.

The occurrence of the hemiplegia took place at the following dates. In the first week no cases, in the second 9, in the third 20, in the fourth to sixth 11. In 14 cases where no exact date is given it is said to have developed in convalescence, in 11 cases no time is given. Recovery took place in 43, death in 18; in 4 no details were given.

Autopsies were held in 15 cases.

Hæmorrhage was found in 1.

Thrombosis in 2.

Embolism in 10.

Embolism and thrombosis in 1.

Sclerotic atrophy of one hemisphere in 1.

In all the cases where details have been given, the initial faucial attack was severe. Albuminuria and ordinary diphtheritic paralysis were present in a large number. Complete recovery was rare. Levi (12) instances only 4 such cases. Almost always contractures and atrophy have supervened in the paralysed limbs. Hemichorea, athetosis, and idiocy are mentioned by Slawyk as sequelæ. Sensibility in most cases was intact. In this respect Rosenthal's case, just quoted, was a remarkable exception. It must be remembered, however, that most of the cases occurred in young children, in whom an accurate estimation of disturbance of sensibility is impracticable.

As Baginsky (13) has clearly shown, hemiplegia differs from all the other palsies of diphtheria in being primarily a vascular

lesion. Thrombosis, embolism, and hæmorrhagic encephalitis are the respective pathological states which give rise to the condition. The nervous system is not primarily attacked as in the common forms of diphtheritic paralysis, but secondarily affected by damage to its blood-supply in one of the three ways mentioned. Slawyck accentuated the fact that a clinical distinction between embolism, thrombosis, and hæmorrhage is impossible, so that in the present case the exact nature of the cerebral lesion cannot be determined. From the definite evidence of cardiac mischief, the probability is in favour of embolism being the cause, as in 10 out of 15 recorded cases in which an autopsy was held. Emboli in malignant diphtheria may be met with in other sites than the cerebral circulation. In Abercrombie's case (14), infarcts were found in the kidneys and spleen. A fatal case of pulmonary and femoral embolism was reported by Poupow (15). Marfan (16) recently narrated at the Société de Pédiatrie at Paris the history of a case of embolism of the abdominal aorta during convalescence from malignant diphtheria. Precocious palatal palsy, enlargement of the liver, and alteration of the normal cardiac sounds preceded the onset of symptoms as in the case under discussion.

Among other features of interest in this case are the early onset of palatal palsy and enlargement of the liver characteristic of the malignant forms of diphtheria (17), the association of characteristic diphtheritic palsies concurrent with the hemiplegia, the triviality of the serum manifestations in spite of the massive doses of anti-toxin (18), and the alternate administration of belladonna and bromide of potassium in cardiac vomiting suggested to me first by Dr Garratt (19).

I am indebted to Dr J. E. Beggs, medical superintendent of the Grove Fever Hospital, for permission to publish this case.

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## Abstracts

### ANATOMY.

**THE AFFENSPALTE AND THE OCCIPITAL OPERCULUM IN (340) THE HUMAN BRAIN.** (Üeber die Affenspalte und das Operculum Occipitale des menschlichen Gehirns.) E. ZUCKERKANDL, *Arb. a. d. neurol. Inst. a. d. Wiener Universität*, 1905, pp. 207-242, 14 text-figures.

THIS is Professor Zuckerkandl's reply to my criticism (in this Review, Vol. ii., No. 6, June 1904, pp. 443-448, and elsewhere) of his views on the morphology of the occipital region of the cerebral hemisphere. The specific remarks and criticisms which I then made with reference to his earlier memoirs apply with equal force to the present contribution to the discussion: all the arguments employed in the work under consideration have been answered in my preceding criticism. Nevertheless, it is useful to consider once more the general bearing of his statements and the logical consequences of the adoption of his views.

The sum and substance of his contention can be briefly stated. In such a brain as that of a Macaque there is a great cleft between the occipital operculum and the "primary parietal lobe," by which term Zuckerkandl distinguishes the superior parietal lobule with the angular gyrus. In this great cleft, which he calls "Affenspaltegrube" (*fossa parieto-occipitalis lateralis*), there are three annectant gyri (*Übergangswindungen*) separated by limiting sulci, all hidden under the occipital operculum; and, in addition, the *fissura parieto-occipitalis* opens into the "fossa." The term "Affenspalte" is strictly reserved (by Zuckerkandl) to the

superficial cleft, *i.e.* the line where this fossa opens on the surface; it is, in fact, merely the mouth of the cleft between the occipital operculum and the "primary parietal lobe."

This narrowly circumscribed definition opens the way for an infinite variety of arbitrary and pedantic distinctions between the conditions found in various apes. The true "Affenspalte" would then be found with any degree of constancy only in Macaques and Cercopithecues, for the retraction of the upper part of the occipital operculum in the Baboons, and more especially in the Semnopithecues, Gibbons, Anthropoid Apes, and Man separates the occipital operculum from the superior parietal lobule, and so destroys the upper part of the Affenspalte, if this is recognised as being merely the cleft between these *apposed* convolutions. According to Zuckerkandl, in proportion as the occipital operculum becomes rolled back and the floor of the "Affenspaltegrube," *i.e.* the annectant gyri, becomes exposed, more and more of the "Affenspalte" becomes destroyed, *i.e.* vanishes, because its lips are no longer in apposition.

Such a definition utterly ignores the only factor of any importance in the interpretation of the morphology of this cortical area, namely, the causal element in the formation of the sulcus lunatus in all its forms, whether or not we agree to call it "Affenspalte" or "Affenspaltegrube."

In all primates (excepting man in rare instances) a part of the cortical area containing the stripe of Gennari—area striata *mihi*—extends from the mesial surface round the occipital pole on to the lateral aspect of the hemisphere. In the larger New World apes, in all the Old World apes, and in most cases in man, a furrow develops at the anterior border of this area striata. This is the sulcus lunatus. As the sulcus deepens, it does so at the expense of the cortex in front of the area striata; in other words, the area striata pushes itself forward and forms the superficial layer only of the occipital operculum which is thus formed. The forward projection of the area striata is clearly the causal factor which produces the fossa. The sulcus lunatus is like the trough in front of the advancing wave formed by the area striata; it has this relationship in all brains where it occurs. And as that cleft in the Macaque's brain, which Zuckerkandl (in common with other writers) calls "Affenspalte," marks (in most cases with mathematical exactness) the cephalic limit of the area striata on the surface of the brain, it is surely not unreasonable to apply the same name to the furrow which presents exactly the same relationship to the identical cortical area in the brains of the higher apes and man? Though at first glance Zuckerkandl's definition is plausible and logical, when we come to examine it we find it not only useless but confusing, because it distracts the

attention from the causal relationship which exists between the area striata and the furrow in question. His elaborate arguments to convince his readers that the sulcus lunatus is not the Affenspalte would, if successful, destroy the value of the only trustworthy guides to the interpretation of the occipital sulci, and in fact of all cerebral furrows, namely, their relationship to cortical areas of distinctive structure.

The caudal lip only of the sulcus lunatus can be regarded as homologous in different brains: the constitution of the floor and anterior lip of the furrow is subject to a considerable range of variation. If, therefore, we attempt to employ the word *homology* in the strict sense in which it is applied in reference to *structures* (rather than mere *folds* of these structures), there can be no doubt that no such homology exists in the case under discussion. The area striata may be strictly homologous in two brains, but the furrow in front of it cannot be so exactly identical if it is deeper in one brain than the other. But if such criteria be applied to cerebral sulci, the utmost chaos must be the only result, because it can happen only in the rarest cases that the walls of two sulci, even in two halves of the same brain, are formed of exactly homologous cortical areas. In speaking of the homologies of sulci, then a certain degree of latitude is necessary in all cases. Yet, I believe, very few sulci present a more definite and exact relationship to an easily recognisable cortical area than that of the sulcus lunatus to the area striata; if, then, we are permitted to use the term homology in reference to furrows of the cerebral hemisphere at all, the sulcus lunatus is one of the few furrows in regard to which the term can be used with justification.

No furrow presents a more definite relationship to two well-defined cortical areas than the rhinal fissure, formed as it is by a folding along the boundary line between the pyriform lobe and the neopallium. If, however, we take two hemispheres, one with a rhinal fissure 1 mm. deep and the other with a rhinal fissure 10 mm. deep, the superficial lips of the latter will be 18 mm. further apart than those of the former, and therefore the furrows are not homologous, according to Zuckerkandl's argument.

But to take a more striking example, *i.e.* of the "calcarine fissure," concerning which Zuckerkandl has written a good deal in his recent memoirs—Zuckerkandl has not hesitated to apply this term and to suggest a definite homology between the various furrows called by this name in a whole series of simian and human brains. Now, the furrow so named in the ape is usually a complete infolding of the mesial part (only) of the area striata, whereas the furrow called "calcarine fissure" in the human brain may (and usually does) consist of an anterior part, one of whose lips is area striata and the other non-striate cortex, and the rest a mere depression in



(and not a complete infolding of) the *whole* area striata, *i.e.* the region which in the ape is partly mesial and partly lateral. In other words, the caudal part of the human "calcarine fissure" is often strictly homologous with the sulcus, which, in the ape, Zuckerkandl calls not "calcarinus," but "occipitalis." Thus, the constitution of the lips of the simian and human "calcarine fissures" may be utterly different. The mode of criticism which Zuckerkandl applies to my identification of the Affenspalte is much more destructive to his own homologisation of the calcarine and other (*e.g.* Sylvian) furrows of the brain.

If, however, we admit the validity of the criteria usually recognised for identifying cerebral sulci, it will be found that the human brain in most cases possesses a furrow, the sulcus lunatus, which has at least as much right to be considered the "homologue" of the Affenspalte of an ape's brain, as the pairs of furrows called central, calcarine, and Sylvian respectively, have in the two brains.

The greater part of Zuckerkandl's memoir is devoted to a detailed account of the variations in form of the occipital operculum and the annectant gyri in the human brain. This does not lend itself to condensation, and the reader must consult the original for it.

In the last two pages of his memoir he attempts to discount the value of the stria Gennari as a guide to the identification of sulci; but it is clear from his statements (especially that of the last paragraph) that he cannot have examined many *fresh* ape brains for this stria. After examining several hundred ape brains in the fresh state, I can state that, excepting in the anthropoid apes (in many cases) and man (in most cases), and rarely in the baboon, the stria Gennari can be clearly seen (with the naked eye) in sections stopping sharply at the free edge of the occipital operculum.

G. ELLIOT SMITH.

**EXPERIMENTAL INVESTIGATIONS ON THE ORIGIN OF THE (341) VAGUS NERVE AND THE CENTRAL TERMINATIONS OF THE SENSORY VAGUS FIBRES ARISING IN THE PLEXUS NODOSUS, AND ON THE COURSE OF THEIR SECONDARY TRACT.** (Experimentelle Untersuchungen über den Ursprung der dem Plexus nodosus entstammenden sensiblen Vagusfasern, sowie über den Verlauf ihrer sekundären Bahn.) KOSAKA and YAGITA, *Okayama Igakkwai Zasshi*, N. 188, August 31, 1905.

THIS is a preliminary communication of the results of investigations which are not yet complete. The authors confirm the

recent previous observations as regards the motor nature of the dorsal nucleus of the vagus nerve. They regard it as the motor centre for the stomach, the smooth muscles of the trachea and the bronchial tubes. The nucleus is not connected with the glosso-pharyngeal nerve, nor with the vagus roots of the opposite side. The nucleus ambiguus, as previously stated by Bunzl-Federn, was divided into two parts, the lower consisting of scattered cells, and the upper of closely aggregated cells. The lower portion is the centre for the innervation of the laryngeal muscles; the upper innervates the pharynx and œsophagus, as well as the crico-thyroid muscle. There is no crossed origin from the nucleus ambiguus of the opposite side.

The plexus nodosus (the ganglion cervicali) gives off about a fourth of its nerve fibres to the abdominal viscera. It is probably also connected with the lungs; it is not connected with the sensibility of the pharynx. After section of the vagus above the plexus nodosus, degenerated fibres could be traced into the solitary bundle, and thence into the ventro-lateral angle of the nucleus cuneatus.

The secondary tract of the sensory vagus is necessarily more difficult to establish owing to the impossibility of isolated destruction of the nucleus of the fasciculus solitarius. The degenerations found after destruction of the fasciculus solitarius were ascending degeneration in the median fillet of the same, and of the restiform body of the opposite side, and in both anterior columns of the cervical cord, where they appear to enter into the anterior cornu. Further details are given as regards the relation of the tract to the pre-dorsal and dorsal longitudinal bundles on both sides. The authors regard this "solitario-spinal tract," as they term it, as an important tract connecting the sensory vagus with the anterior roots of the spinal cord, and as probably playing an important part in respiration. It is open to question, however, whether this tract is really an indirect continuation of the sensory vagus fibres. It must be almost impossible to destroy this without interfering with some of the lower connections of the nucleus of Deiters, etc.

From experiments involving extirpation of the lower lobes of the right lung, the authors found a degeneration of the plexus nodosus, but none in the vagus above this or in the nucleus ambiguus. They infer that the smooth muscles of the lungs are innervated not from the vagus, but from the sympathetic, although they do not exclude an indirect action of the motor vagus upon the bronchi.

The paper is of value, and should be read in the original. It will, it may be hoped, soon be followed by an account of the completed research.

ALEXANDER BRUCE.

**FURTHER RESEARCHES UPON THE GENESIS OF THE NERVE****(342) CELLS.** (*Ulteriori ricerche sulla genesi delle cellule nervose.*)FRANCESCO CAPOBIANCO, *Ann. de Neurolog.*, 1905, f. i.-ii., p. 50.

THE view that the nerve cells are of multiple origin was first advanced in 1899 by Fragnito, on the ground of observations made upon the bulbar and cerebral elements; in the following year, Capobianco brought forward similar evidence derived from observations upon the spinal cord and intervertebral ganglia. This view has since been supported by the results of the investigations of several workers, but it has not as yet been generally accepted. The author has therefore put on record some further observations which bear out his previous conclusions.

He has studied kitten embryos of from 3 to 5 c.m., and the human foetus at the third month. With the aid of a series of very clear illustrations, made from preparations stained with hæmatoxylin and scarlet, he describes the successive stages in the development of the nerve cells from the neuroblasts in the spinal ganglia and cord. This transition takes place at a definite period and is accomplished in a comparatively short space of time. Small groups of neuroblasts become approximated, their protoplasm fuses into a single mass, and certain of the nuclei undergo regressive changes and finally disappear. Not only the body of the nerve cell but also its processes are formed by this fusion of neuroblasts. The author has also investigated the numerical relation between the neuroblasts and the nerve cells in the spinal ganglia. In one instance he found it to be as 2·83 to 1, and in another as 3·2 to 1. As there is evidence that the small cells result from the union of only two neuroblasts, these figures harmonise with the view of the multiple origin of the nerve cell.

W. FORD ROBERTSON.

**PHYSIOLOGY.****COMMUNICATION UPON THE INFLUENCE ON WORK OF****(343) SIMULTANEOUS SENSORY STIMULI.** (*Note sur l'influence de quelques excitations sensorielles simultanées sur le travail.*)FÉRÉ, *Comp. rend. des séances de la soc. de biol.*, June 10, 1905, T. lviii., p. 979.

IN a previous paper Dr Féré has shown the effect upon work (as registered by the ergograph) of successive sensory stimuli; in this communication he deals in a similar way with simultaneous stimuli. The method of experiment is as follows:—Two or more of the senses are appropriately excited for a period of 20 seconds;

one ergogram is written immediately, and a second after 18 minutes' rest—the time normally required for the complete restoration of the muscle. I quote one or two of the figures:—

Sense excited.	Work in kilogrammetres.	
	1st Ergogram.	2nd Ergogram.
Touch and smell . . .	·75	5·22
Touch, smell, and taste . .	·45	2·40
Touch, taste, hearing . .	·57	3·36
Touch, smell, taste, sight .	·48	1·95

The normal ergogram being 9·60, the experiments show that the effect of the stimuli is markedly to decrease the amount of work done, not only in the first ergogram, but in that written after the period of rest. Dr Féré does not say whether his experiments were conducted on more than one person, nor whether his figures are derived from an average, but his results are undoubtedly very striking. MARGARET DRUMMOND.

**ON THE INFLUENCE OF TOXIC AND MEDICINAL SUB-**  
**(344) STANCES DURING REST AND AFTER WORK.** (Note sur  
 l'influence de substances toxiques et médicamenteuses au repos  
 et après le travail.) FÉRÉ, *Comp. rend. des séances de la soc. de  
 biol.*, June 10, 1905, T. lviii., p. 979.

In this paper Dr Féré gives, by aid of the ergograph, measurements of the different effects of certain drugs (bromide of potassium, veronal, and extract of valerian), according as they are taken after rest or after work. One experiment will serve as example. Five grammes of bromide of potassium were taken, with the result that the ergogram written five minutes later showed only 3·36 kilogrammetres of work; while after eight minutes' rest the work had fallen to ·84. When the same experiment was tried, after work had already been done, the first ergogram showed 12·15 kilogrammetres.

MARGARET DRUMMOND.

**(345) PAIN AND FATIGUE.** (*Douleur et fatigue.*) *Ibid.*, July 1, 1905

CONTINUING his study of the effects of sensory stimuli on work, Dr Féré here enquires what happens when the sensations are so acute as to be termed pain. The forearm was placed in Mosso's ergograph, and to it by means of Bloch's sphygmometer painful pressure was applied. A tracing was written immediately after the removal of the stimulus, and a second after eighteen minutes' rest. It was found that when the pressure is slight and short,

a scarcely perceptible increase of work results (9.69 instead of the normal 9.60), and the muscle is restored in the normal time. As the pressure increases in strength and duration the power of work diminishes and fatigue persists. Thus if the pressure used in the experiment referred to above is prolonged from 20 to 40 seconds, the ergograms decrease to 2.58 and 2.47. Increase of pressure produces a similar effect.

If, however, the painful stimulus is applied to a muscle when the muscle is already fatigued, then it acts as an excitant increasing the amount of work done. For example, the conditions which before resulted in 9.69 kilogrammetres of work will in the new conditions give 12.48. But as pain is increased in duration or intensity, it loses its power of acting as an excitant.

The general conclusion is that painful stimuli act in the same way as disagreeable or multiple stimuli, but they give rise to a more lasting fatigue.

MARGARET DRUMMOND.

**NEW EXPERIMENTS ON THE INFLUENCE OF SOUP ON (346) WORK.** (*Nouvelles expériences sur l'influence du bouillon sur le travail.*) FÉRÉ, *Comp. rend. des séances de la soc. de biol.*, July 29, 1905, T. lxx., p. 233.

By a former series of experiments Dr Féré was led to the conclusion that soup owes its properties as an excitant mainly to its action as a sensory stimulus. The present series was undertaken under somewhat different conditions to act as a check upon the previous conclusion.

Two sets of experiments were performed. In the first, 20 c.cm. of soup were held in the mouth for 20 seconds; in the second, 100 c.cm. were swallowed in three or four mouthfuls; in each case two ergograms, with the usual eighteen minutes' rest between, were written. The figures, which show that the former set of conditions is productive of the greater increase of work, confirm Dr Féré's previous opinion that the excitation is mainly due to the sense stimulation. It is shown also that in both cases the temperature of the liquid is of considerable effect; an increase of temperature increasing the stimulating action.

MARGARET DRUMMOND.

**NOTE ON YAWNING.** (*Note sur le bâillement.*) *Ibid.*, July 1, (347) 1905, T. lxx., p. 11.

That yawning coincides with or is followed by a real depression of voluntary activity was noted by Dr Féré in the course of a

series of ergographic experiments which he had occasion to make with his eyes shut. Twenty distinct efforts were made, separated from one another by one minute's rest. The darkness had a depressing effect, which brought on unwonted attacks of yawning, which, however, never appeared during the work, but always during the periods of rest. When the yawning appeared during the last half minute of rest, it was followed by a very marked decrease of work.

MARGARET DRUMMOND.

### PSYCHOLOGY.

**CONTRIBUTION TO THE PSYCHOLOGY OF TWINS.** (Contribution à la psychologie des jumeaux.) FÉRÉ, *Comp. rend. des séances de la soc. de biol.*, July 29, 1905.

IN this brief paper Dr Féré describes a case of what may be called an interchange of character. The subjects were twin girls eighteen years of age. The father and mother were 48 and 44 years of age respectively; heredity was good, no previous case of nervous or mental trouble being recorded; this was the first case of twins in the family. The girls differed markedly in both physical and mental characteristics: the one was fair, tall, and thin; the other dark, shorter, and of fuller development; their features were quite unlike. Towards their half-brother, three years older than they, the fair twin, who was the more demonstrative of the two, manifested an incorrigible antipathy; the dark one, an apathetic child, showed no initiative, but suffered his caresses. The twins had the usual ailments of childhood, but at different times. But a striking coincidence was shown in the phenomena preceding menstruation, which were of the same nature in both cases, and occurred at precisely the same time. On the day after menstruation was established the mental characteristics of the two girls were abruptly interchanged. The dark one became more frank and demonstrative, attending classes for singing and dancing (which she had always previously disliked) with pleasure, and she could no longer bear the sight of her brother. The fair girl, on the other hand, took up her sister's part, becoming apathetic and indifferent, speaking only enough to express her wants, and showing no antipathy towards her brother. These characteristics so strangely interchanged are becoming accentuated in the two sisters.

MARGARET DRUMMOND.

**PATHOLOGY.**

**CONTRIBUTION TO THE PATHOLOGICAL HISTOLOGY OF THE**  
 (349) **GASSERIAN GANGLION.** (Beitrag zur pathologischen  
 Histologie des Gasser'schen Ganglions.) CAMINITI, *Arch. f.*  
*klin. Chirurgie*, 1905, lxxvii., p. 901.

AFTER referring to his earlier work ("Recherches sur l'anatomie chirurgicale du ganglion de Gasser"), published in 1904, in which he had directed his attention chiefly to the micro- and macroscopical anatomy of the ganglion, Caminiti turns to a consideration of its pathology, the uncertainty and lack of completeness of which he attributes to defective histological investigation.

Changes in the ganglion, partly and chiefly in the connective tissue, partly in the cells, have been found by D'Antona, Krause, Sanger, Coelho, Pettana, Barker, Spiller, Schwab, Keen, and Coenen. By analogy with other changes in the nervous system, Caminiti thinks these alterations may in many instances explain the neuralgia, but that they should by no means be regarded as a characteristic sign of it, and consequently as a constant symptom in every case of painful tic.

He then raises the following questions:—

1. Whether the changes described were constant in appearance upon extirpation of the Gasserian ganglion in consequence of trigeminal neuralgia.
2. Whether the changes described were identical in every ganglion extirpated on account of trigeminal neuralgia, and submitted to histological examination.
3. Whether the changes that were found were present only in cases of trigeminal neuralgia.
4. Whether the changes were the same, either if the operation was done initially upon the ganglion, or if previously peripheral resections had been performed upon branches of the fifth nerve.

Caminiti answers the first question by pointing out that the greatest number of extirpated ganglia were not histologically examined, and among the few that were so examined were some in which no trace of any change was present (Monari, Schwab, Richard, Poirier, Coenen).

Concerning the second question, he remarks that many showed evidence of change, varying in degree and nature, in the cells only; others in the connective tissue; others again in both elements of the ganglion, but in varying degree. Nor has there existed any basis of complete agreement concerning the nature of these changes, D'Antona designating them sclerosis of the ganglion; Coelho, yellow degeneration; Sanger, hypertrophy and

hyperplasia of the connective tissue with amyloid degeneration ; Keen, inflammatory infiltration ; Spiller, endothelioma.

Regarding the third question, Caminiti remarks that the same changes were shown in cases in which no neuralgia was present, and that in cases of trigeminal neuralgia there were found lesions of other parts quite unconnected with the ganglion.

In respect of the fourth question, he recalls that Coenen, upon histological examination of fifteen cases in which the ganglion was extirpated for trigeminal neuralgia by Lexer in Bergmann's clinic, found changes in thirteen cases in which, antecedent to the present operative interference, operations had been carried out at more or less remote periods on the peripheral branches. Whilst, in the two cases in which no operation had been previously undertaken, he found the ganglion normal. Lexer, from animal experimentation, reached the same conclusion as Cushing and Barker had done, viz. that after peripheral operations upon the fifth nerve, changes are found at varying periods in the nerve cells of the ganglion.

Caminiti concludes as the result of his investigation that trigeminal neuralgia depends in some cases directly upon a lesion of the ganglion ; in others it arises indirectly as a consequence of pressure, which is provoked by tumours, meningitis, cranial distension, etc. Finally, it may develop as a sequel to peripheral disturbances or in consequence of a general infection. In the latter case the extirpation of the ganglion and its histological investigation would yield a negative result.

He then proceeds to discuss the histology of the ganglion as found by himself in healthy individuals of 35 years and older, and compares it with the so-called pathological changes described by others. He finds, as a result of his observations, a difference in the cells in respect of size and form ; actual differences in the intensity of staining ; partial radiations in the body of the cell of the chromatic substance, the arrangement of which in some cells is in fine, in others in coarser granules ; slight thickening of the chromatic substance, sometimes around the nucleus, sometimes around the periphery of the cell ; sometimes an exchange of the place of the nucleus from the centre to the periphery ; in most cells the constant presence of yellow pigment in varying quantity and different arrangement ; cells with scanty remnants of protoplasm without nucleus ; and, finally, capsules either without protoplasm or sometimes with an accumulation of nuclei in the place of the protoplasm.

These observations, as he points out, frequently agree perfectly with those which have been described by others as pathological in the ganglion. He then discusses at some length the differences in the size of the ganglion cells, the question of the cell-stainings,



the differences in the arrangement and quantity of the chromatic substance, the occurrence of the empty capsules, the vacuolated and the non-nucleated cells, and the presence of the yellow pigment.

He concludes that the numerous changes in the nerve cells described in the Gasserian ganglion are entirely non-characteristic of trigeminal neuralgia, and belong only to the realm of possibility; that many of them are normal accompanying phenomena—and normal certainly in the most general sense—of advanced age and old age; many others are met with in the most diverse diseases which attack the nervous system directly or indirectly. He thinks, however, that the changes described as occurring in the fibres and connective tissue of the ganglion are unique; that they terminate in involution and sclerosis; and are capable, when they exist, of supplying a sufficient warrant that the neuralgia has had its seat in the Gasserian ganglion. They would also explain the neuralgia and the cell lesions which have been described, which would thus be a secondary phenomenon resulting therefrom.

DONALD ARMOUR.

**ON THE ACTION OF VENOMS OF DIFFERENT SPECIES  
(350) OF POISONOUS SNAKES ON THE NERVOUS SYSTEM.  
IV.—Venom of *Daboia Russellii*. GEORGE LAMB and  
WALTER K. HUNTER, *Lancet*, Sept. 23, 1905, p. 883.**

THE action of *daboia* venom varies with the amount given. In larger doses the symptoms set in immediately, there being restlessness, laboured respiration, general convulsions, and death within a very few minutes. On post-mortem examination an extensive intra-vascular thrombosis is to be found, but affecting principally the pulmonary arteries; and this thrombosis doubtless accounts for the symptoms and is the cause of death. But with small doses of the venom the coagulability of the blood is not increased but is lessened; so much so that by injecting a small preliminary amount of the venom, the larger dose can be given later without there being any intra-vascular clotting whatever. Death in such cases is manifestly not due to thrombosis, and L. Rogers is of opinion that it is the result of a vaso-motor paralysis of central origin. But confirmation of this, by finding degenerative changes in the cells of the vaso-motor nucleus, could not be hoped for in animals dying within a few minutes after receiving the poison. *Daboia* venom, however, also kills in smaller doses, and death may then be delayed for from one hour to several days. But the symptoms in these cases point rather to a hæmolysis than to an action

on the nervous system. This is confirmed by an histological examination of the central and peripheral nervous system in nine monkeys killed with daboia venom, and dying at periods varying from four to sixty hours. In all of these the appearances were quite normal. In four of them serial sections were made through the vaso-motor "area" in the medulla (in two of the cases the sections were transverse and in two longitudinal through the lateral nucleus), but without revealing any unequivocal changes in the ganglion cells of this area. These observations, therefore, give no support to the suggestion that the symptoms in the more chronic daboia poisoning are dependent upon a vaso-motor paralysis of central origin.

W. K. HUNTER.

**AN INVESTIGATION ON THE REGENERATION OF NERVES  
(351) WITH REGARD TO THE TREATMENT OF CERTAIN  
PARALYSES.** BASIL KILVINGTON, M.S., M.D., Melbourne,  
*Brit. Med. Journ.*, April 29 and Sept. 16, 1905.

THIS interesting paper gives an account of a research undertaken for the purpose of determining whether existing methods of nerve suturing, where the peripheral end of a damaged nerve had to be united to another nerve trunk, could be improved upon, and why failure not infrequently occurred.

In the first paper (April 29) the author demonstrates experimentally on dogs that if the central end of one nerve be sutured to the peripheral ends of two divided nerves it was impossible to obtain, by the longest period of education, perfect co-ordination, and to prevent antagonistic muscles, enervated as they now might be from the same nerve cells, interfering with any definite movement. This is due, the author believes, to the splitting of axis-cylinder processes, with the result that one half may go to a muscle which antagonises the action of the muscle which the other in part supplies.

In the second part of the paper (Sept. 16) the following experiments were described. The internal popliteal nerve was divided in dogs in such a way that one half of the original nerve was continuous with the *whole* internal popliteal, while the remaining and smaller half was united to the external popliteal—the internal popliteal thus innervating both internal and external popliteals. The author urges that the greatest care should be taken in dividing the internal popliteal trunk to prevent a lesion of any of the funiculi, thus preventing splitting of existing axis-cylinders which could by any chance enable the divisions of one axis-cylinder reaching antagonistic muscles. Electrical tests were applied, months after the operation, in successful cases, and the

results were satisfactory, while co-ordinated movements appeared to be largely dependent on the care taken to obviate any chance of one nerve cell by division of its axis-cylinder process supplying antagonistic muscles. In one experiment an unsuccessful result was found to be due to fusing of the two nerves at the site of operation, and probably therefore an interchange of axis-cylinder processes.

In a second series of experiments into gaps or slits in the internal popliteal at different levels, both the central and peripheral ends of the divided external popliteal nerve were inserted and sutured, a portion of the nerve being excised. In these cases, part of the internal popliteal between the slits was used as a channel of communication, but axis-cylinders from the internal popliteal naturally found their way into the peripheral part of the external popliteal, and similarly from the external popliteal central end fibres passed into the internal popliteal. This method of utilising the central end of the divided nerve where it is impossible to unite it directly to its own peripheral end is not to be commended, unless modified as described below. It is better to split the healthy nerve trunk with the greatest care and to take a suitable part of it for uniting by suture with the peripheral end of the damaged nerve. This prevents stimuli overflowing into the wrong channels. It may, however, in certain cases be possible to utilise the central end of the damaged nerve, as well as the peripheral, but the separation of the portion of the healthy nerve trunk used for the operation should be continued up to and beyond the level of the slit where the central end of the damaged nerve is inserted. By this means the portion of the healthy nerve so separated acts as a graft down which nerve fibres will in time pass from the central end of the damaged nerve itself. The paper, if erring on the side of brevity and, therefore, want of clearness to those not versed in the subject, is of great interest.

ROBERT A. FLEMING.

**DEGENERATION OF NERVE-CELLS OF THE RABBIT'S  
(352) SUPERIOR CERVICAL GANGLION AS THE RESULT  
OF INTERFERING WITH THEIR BLOOD SUPPLY. IVOR  
TUCKETT, *Journ. of Physiol.*, xxxiii., 1, Sept. 8, 1905, p. 77.**

THE superior cervical ganglion of the rabbit was dissected out from the surrounding tissues, the cervical sympathetic nerve left intact, but the vessels from the internal and external carotids divided. Degeneration was found to start in the cells of the ganglion immediately, and to be usually proportionate in extent

to the time that had elapsed between the operation and the final fixation in corrosive sublimate. Three main stages are distinguished:—

First stage.—The nucleus stains uniformly with methylene blue, the nucleolus is still deeply stained. Both nucleus and cell are shrunken, and there is a markedly less stained area of protoplasm around the nucleus.

Second stage.—The nucleolus is lost. Nucleus and cell are more shrunken, and the cell body begins to stain round the periphery with methylene blue instead of eosine.

Third stage.—There is an inversion of the staining reaction; the shrunken nucleus stains with eosine and the protoplasm of the cell more or less with methylene blue.

Finally, nucleus and cell merge into one.

If a cervical ganglion is excised and placed in the peritoneal cavity, degeneration takes place more slowly, probably owing to a better supply of lymph in that situation.

PERCY T. HERRING.

**A CONTRIBUTION TO THE PATHOLOGY OF THE SO-CALLED (353) RAYNAUD'S DISEASE, OR SYMMETRICAL GANGRENE.**  
(Beitrag zur Pathologie der sogen. Raynaud'schen Krankheit oder symmetrischen Gangrän.) Dr J. M. BELKOWSKY, Cleveland, Ohio, *Neurolog. Centralbl.*, Sept. 16, 1905, p. 836.

In a well-marked case of Reynaud's disease, with gangrene of the fingers, toes, nose, and ears, the author found on microscopical examination of the spinal cord a general condition of arteriosclerosis of the intramedullary vessels. At different levels of the cord associated with the larger of these sclerotic vessels there is a diminution in the size of the grey matter, and of the number of ganglion cells present. There is also a marked degeneration of fibres about the whole periphery of the cord.

In the *lumbar cord* the same changes are present, but more marked chromatolysis and pigmentation in the ganglion cells, the cells of Clarke's column alone remaining well stained.

The *sciatic nerve* is much smaller than usual, and shows numerous degenerated fibres.

COLIN K. RUSSEL.

### CLINICAL NEUROLOGY.

**THE NATURE OF TABES.** Sir WM. GOWERS, *Brit. Med. Journ.*, (354) July 8, 1905, p. 57.

In the course of a clinical lecture on the nature of tabes, attention is called to what Gowers regards as the essential symptom of the

disease. The loss of tone is not the result of the impairment of the motor nerves or centres; it may occur without any change in the cutaneous sensibility. It can therefore only be ascribed to the impairment of afferent muscle nerves, the peripheral sensory nerves of the muscles. Wherever there is marked ataxy the sensitiveness of the muscles will be found to be impaired or lost. Strong tension in health causes considerable discomfort, but in tabes is unperceived. By a simple expedient the insensitiveness of the muscle to the contraction excited by the faradic current may be demonstrated. The skin of the calf is pinched up between two small electrodes, and a faradic current of adequate strength is passed between them. Only the tingling sensation of the current is felt. If now the electrodes be separated, keeping them in contact with the identical points of the skin so that they rest on the muscle, this will contract strongly, but the tabetic will observe nothing beyond the initial skin sensation. The muscular tone is a reflex adjustment of the muscles dependent on afferent impulses which chiefly come from their nerves. Other sensory impulses from the skin may play a subordinate part in the reflex process, but the features of tabes show that these are subordinate to the influence from the muscles. Tone is essentially a muscle reflex process. In the derangement of this reflex mechanism we may perceive one cause of the ataxy of tabes.

Gowers has repeated the statistical studies of Erb, in which the percentage of cases of tabes in which a definite history of a primary chancre could be obtained was compared with that where a definite history of chancre could be obtained from patients suffering from undoubted syphilitic rashes. In such cases of syphilitic skin lesions collected at University College Hospital, the primary disease was only known to have existed in 80 per cent., and this is exactly the same proportion that was found in cases of tabes observed in private practice. Another noteworthy proof of the dependence of tabes on syphilis is offered by the invariable presence of signs of hereditary syphilis in cases of "juvenile tabes."

Tabes cannot be explained by conceiving that an early toxine simply lessens the vital endurance of the nerve elements. The facts of rapid development and rapid increase, even after years, compel the conviction that the morbid agent is present at the time. Equally incompatible with the theory of lessened vital endurance is the remarkable degree of recovery sometimes met with. Hence it has been suggested that some other agent of unknown nature co-operates with that which syphilis produces.

F. GOLLA.

**A CASE OF POLIOMYELITIS ANTERIOR ACUTA WITH  
(355) OCULO-PUPILLARY SYMPTOMS. (Über einen Fall von  
Poliomyelitis anterior acuta mit oculopupillaren Symptomen.)**  
CLOPATT, *Deutsch. med. Woch.*, Sept. 21, 1905, p. 1495.

IN this case of poliomyelitis affecting a little child two years and four months old, the chief incidence of the disease was on the left arm, which was helpless except for some slight movement of the fingers. Further, the upper eyelid on the left side drooped somewhat, and the left pupil was smaller than the right, though the range of its contraction to light exceeded that of the other. Opinion as to there being any enophthalmos was reserved. At the end of four months there was a considerable return of power in the arm and the inequality of the pupils had disappeared, but the ptosis persisted.

The evidence seemed to indicate that the cilio-spinal centre of Budge had been involved in the lesion, causing a paralysis of the dilator pupillæ and the orbital muscle of Müller. In spite of the frequency of cervical and dorsal poliomyelitis, it is curious how seldom this centre is injured, at least if one is to judge from the rarity of similar cases in the literature. S. A. K. WILSON.

**INFANTILE NUCLEAR ATROPHY. (Über infantilen Kern-  
(356) schwund.)** GIERLICH, *Deutsch. med. Woch.*, Sept. 14, 1905, p. 1462.

TO this rare condition attention was called by Möbius in 1888, and the view advanced that it was due to an aplasia of the motor cranial nerve nuclei, though the agenesis might conceivably also have been in nerve trunks or muscles. Within recent years one or two autopsies have aided in its elucidation.

The case reported concerns a little girl of fourteen years at the time of examination. At birth there was no indication of hereditary syphilis, but from the outset she had been unable to suckle, while saliva dripped from her lips incessantly, and tears from her eyes. When she came under observation, no developmental anomalies were discovered, and no signs of previous rickets. The face was stiff and mask-like, with practically no facial movements. The lower lids were immobile, while the upper contracted very slightly, and both in volitional and expressional action the corners of the mouth moved feebly down and out. No fibrillary twitchings were noticed at any time in the muscles. The whole of the left half of the tongue was furrowed and atrophic, except for a

distance of five millimetres from the tip. On protrusion it deviated markedly to the left. Pupillary reactions were normal, but there existed a complete paralysis of conjugate movement in either lateral direction, both for active and for reflex movement. Convergence, however, was intact.

The fact that there was complete loss of reaction to faradism and to galvanism, except at the corners of the mouth, is very significant, as it indicates the peripheral, *i.e.* lower neuron, nature of the lesion, which the double conjugate paralysis is of help in localising. Its probable site is in the formatio reticularis of the lower part of the pons.

In a case recorded by Heubner there was simple absence of certain motor cranial nerve nuclei, their place being taken by pure glial tissue. Hence atrophy is less accurate a description than aplasia or hypoplasia. Nevertheless this is not a feasible explanation of all the observed cases, and fresh information is desirable.

S. A. K. WILSON.

**PARAPLEGIA FROM FRACTURE OF THE FIRST, SECOND, (357) AND THIRD DORSAL VERTEBRÆ; SEVEN OTHER FRACTURES; LAMINECTOMY; RECOVERY WITH ABILITY TO WALK WITH ASSISTANCE. GUY HINSDALE, *Journ. of Nerv. and Ment. Dis.*, Sept. 1905, p. 562.**

IN this case, as the result of trauma, there was complete motor and sensory paralysis downwards from a line drawn two inches below the nipples; total loss of control over bladder and rectum; loss of reflexes, including knee-jerks and plantars. Laminectomy, performed four days later, showed slight laceration of dura, cord oedematous but not severed; dura was not sutured, cerebro-spinal fluid drained off freely.

During the first two weeks there were convulsive attacks, rapidly increasing in violence, affecting the non-paralysed parts, *i.e.* arms, shoulders, neck, face; no loss of consciousness; difficulty of breathing; spasm of diaphragm; movements of jaw and laryngeal muscles. All these movements subsided as the flow of cerebro-spinal fluid ceased.

Dissociation of sensibility was marked within two weeks; absolute analgesia and thermo-anæsthesia below the line mentioned above, tactile sense preserved over most of the abdomen and legs; profuse perspiration over head and neck.

Knee-jerks returned in a month and soon became exaggerated; ankle-clonus was well established at the end of two months, and it

was present three years after accident. Babinski reflex was noted at the end of a month, and it was present three years later. Bed-sores formed on sacrum and buttock, but healed well. The bladder resumed its normal function three years after accident. Gradual return of power in the legs began at the end of nine weeks, and marked spastic condition of the legs developed, necessitating various tenotomies. Three years after accident, patient could walk in a wheel crutch, and could get from the bed to the crutch with a little help. Dissociation of sensibility was still present nearly two years after the accident, but the analgesia and thermo-anæsthesia were not so absolute, and their upper limit was considerably lower. At the end of three years, tactile sense was quite normal and pain sense was present on many parts of trunk and legs. The tendency to excessive perspiration diminished.

Attention is drawn to these points:—(1) The dissociation of sensibility of the syringomyelic type: this is not an infrequent symptom in injuries of the cord at this level, *c.f.* cases of Kocher, Lloyd, Gordon, etc. (2) The convulsions after laminectomy, where the cerebro-spinal fluid is allowed to escape: a similar case is referred to. (3) Vasomotor disturbances of the head, due to lesion of the three upper dorsal roots.

A. W. MACKINTOSH.

**A CASE OF DIVER'S PARALYSIS, WITH HISTOLOGICAL  
(358) EXAMINATION OF THE SPINAL CORD. W. HALE WHITE  
and F. A. BAINBRIDGE, *Lancet*, Oct. 14, 1905, p. 1101.**

A PROFESSIONAL diver had had several attacks of transient paresis from which he had recovered.

During his last attack he was admitted to hospital suffering from weakness in the legs and cough, the paresis having set in three weeks previously after a long submersion at a depth of 130 feet.

His gait was laboured and there was foot-drop, but no muscular wasting. Sensation to touch and cold was normal; sensation to heat deficient. The knee-jerks were absent, the plantar reflexes were flexor, and the pupils were normal.

He had been addicted to alcohol, and had had scurvy gonorrhœa, and syphilis.

The muscular weakness improved, but he died nine weeks after admission from pulmonary tuberculosis.

The brain and spinal cord were examined. The white matter of the cord was normal. The perivascular spaces in the grey matter of the lumbar region were larger than those in the thoracic and cervical regions. No hæmorrhages were observed.



The case illustrates the power of recovery, for although each attack of paralysis after diving was no doubt due to the escape of gas bubbles into the blood-vessels and tissues of the spinal cord, yet at death three months after the last attack but little change could be seen in the cord; at the most the evolution of gas may have left some permanent distension of the perivascular spaces.

The authors append a micro-photograph of a section through the lumbar region of the cord.

H. DE M. ALEXANDER.

**LUMBAR PUNCTURE.** D. DE BUCK, *Journ. de Neurolog.*, No. 17, (359) 1905, p. 321.

THIS is a long and complete paper of thirty pages with numerous references. He concludes:—

1. The cerebro-spinal fluid is a secretion of the ependyma and especially of the choroid plexus; it passes into the lymphatic circulation either directly by the perivascular spaces which communicate with the arachnoid sac, or indirectly by diffusion. It receives certain products of metabolism from the nervous tissues and participates in the traumatic lesions and morphological changes of these tissues and especially in the meningeal changes.

2. Of its physical characteristics, the colour is the most important. Density and osmotic tension are at present chiefly of scientific interest.

3. In the chemical analysis, the presence of choline and increase in the amount of phosphates are in favour of an organic affection. Albumin, whilst important, may be present in affections which do not show cellular elements in the cerebro-spinal fluid.

Little importance attaches to the degree of the alkalinity, or the amount of chlorides or reducible matter.

De Buck has never found acetone, the diazo reaction, ammonia, or hæmolysines present.

4. The most important examination is that of the cellular elements and the determination of the leucocytic formula.

5. The permeability of the secretory membrane requires further study.

6. The toxicity of the cerebro-spinal fluid is feeble. After injection into animals few symptoms result, but careful examination seems to show that changes in tissue metabolism occur, and these may be important.

7. Bacteriological examination often yields important positive findings in meningitis, but in mental diseases micro-organisms have not by this method been found to play a rôle.

8. Therapeutically, repeated puncture is of value to acute

meningitis, but not of service in mental diseases, even in epilepsy. It may be of service in chronic hydrocephalus, uræmia, traumatic coma, chorea, grave chlorosis with severe cerebral symptoms, whooping-cough, auricular vertigo, and the crises of tabes; also in the eclampsia of pregnancy.

As a means of introducing remedies, the author thinks the method requires serious study and that it may replace sub-dural inoculations.

This paper cannot be abstracted in detail; it contains many personal observations, and a number of observations on the chemical and physical characteristics of the fluid, which will be of much service to any who desire to follow this study.

W. B. WARRINGTON.

**LUMBAR PUNCTURE IN THE INFANT.** AUG. LEY, *Journ. de*  
(360) *Neurolog.*, No. 18, 1905, p. 361.

THIS paper covers much the same ground as that by De Buck, and is also accompanied by a copious bibliography. The writer calls attention to the fact that at birth the cord reaches to the level of the third lumbar vertebra, and that it is only at the beginning of the second year that it has the same relation to the vertebræ as in adult life; hence the importance of tapping the theca as low as possible, namely, in the lumbo-sacral space.

The most useful section of the paper deals with the therapeutic results.

It has been used (1) in chronic hydrocephalus with reported success by many, and has been thought to be a valuable adjuvant to the inunction of mercury; (2) in tuberculous meningitis it has been said to produce a temporary improvement; (3) its repeated practice in the acute meningites is generally admitted to be of considerable service, and it is claimed that by this means of treatment there is less chance of the late affections of the sense organs occurring; (4) in the uræmia of scarlatina, cessation of coma and rapid disappearance of symptoms have been noted; (5) in whooping-cough with cerebral symptoms; (6) Babinski claims that it is useful in auricular vertigo; (7) injection of drugs: Mammack records five cases of the injection of 10 per cent. of lysol in epidemic cerebro-spinal meningitis—one cure and four deaths (*Med. Record*, June 4, 1904).

Tetanus antitoxin is more efficient when injected than when given subcutaneously.

The writer thinks that there is a useful future before this means of medication.

W. B. WARRINGTON.

**THE SURGERY OF TUMOURS OF THE PONTO-CEREBELLAR**

(361) **ANGLE.** (*Zur Operation der Tumoren des Kleinhirnbrückenwinkels.*) BORCHARDT, *Berl. klin. Woch.*, Aug. 14, 1905, p. 1033.

AT Professor Oppenheim's request the reporter operated on a case of intracranial neoplasm which was diagnosed as lying in the angle between the cerebellum and the pons. Death ensued twenty-six hours after the operation. The remarks relative to the surgical technique of such cases are not particularly illuminating.

S. A. K. WILSON.

**FEVER IN HYSTERIA.** (*De la fièvre hystérique.*) CARAMANO, (362) *La Presse Médicale*, Sept. 20, 1905, p. 595.

ALTHOUGH the genuineness of hysterical fever has been denied by Charcot, abundant clinical evidence is forthcoming in its favour. Its onset is characterised by abruptness, and its course by a certain periodicity. After the fever is over, the return to the normal is instantaneous. The intermittent type is represented by a sudden access of fever very frequently towards evening, the duration of which is seldom more than a few hours, while it may attain a height of 104° or even more. There is also an accompanying quickening of respiratory and cardiac rhythm. The differential diagnosis is by no means always easy, and only to be arrived at by resort to the method of exclusion.

Medicinal treatment is of no avail. We can recommend the procedure employed by Vidal, who tells the patient he has ordered a powerful drug for him, of which not more than two tablespoonfuls must be taken daily, as excess would be dangerous. He is further told that if the medicine is efficacious his urine will become blue. The "drug" is of course only a weak solution of methylene blue, to which a few drops of tincture of nux vomica have been added. The end justifies the means.

S. A. K. WILSON.

**AN ACUTE ("POLIOENCEPHALITIS SUPERIOR HÆMORRHAGICA") AND A CHRONIC CASE OF KORSAKOFF'S PSYCHOSIS.** (*Ueber einen acuten, etc.*) S. BOEDEKER (of Schlactensee), *Arch. f. Psych.*, Bd. 40, H. 1.

UNDER the title of polioencephalitis superior hæmorrhagica, Wernicke described in 1881 certain paralyses of the associated movements of the eyes accompanied by delirium, with acute onset and fatal termination, the symptom-complex arising on the

basis of chronic alcoholism. Boedeker and others have shown that this ophthalmoplegia is not a disease *sui generis*, but merely one symptom of a general condition which is intimately related to Korsakoff's psychosis. The termination is not necessarily fatal; the internal as well as the external eye muscles may be affected; most frequently one finds a paralysis of the sixth nerves, and not a paralysis of associated movements. Cases which do not end fatally frequently pass over into a condition identical with that in Korsakoff's psychosis.

Boedeker reports the case of a university professor, extremely sociable, if not exactly alcoholic, who three weeks before admission became weak in the arms and legs, especially on the right side, had left-sided ptosis, double vision, inability to whistle, and in a few days became delirious, failed to recognise his environment and relatives, lived over again his youthful travels, delivered lectures: the condition came on after considerable mental and physical strain, and was accompanied with a bronchitis and temperature of 38.8°. On admission patient seemed dull and exhausted, was completely disoriented, said he was on a geographical excursion, that he had been threatened with revolvers, etc. After a few days patient's physical condition improved rapidly, he was oriented, showed extremely poor retention, gave a fantastic and confused account of his admission and recent doings. Sixteen days after admission patient seemed quite clear, but he continued to relate remarkable histories of the recent past, the incidents being usually real personal experiences, distorted in their time and space relations, and furnished with delirious elaborations. The ptosis had almost disappeared, no paresis of the eye muscles could be objectively demonstrated, although there was still double vision in certain directions. After a few more days patient had completely recovered, and was able later to resume his professorial duties.

The author refers to the initial difficulty of diagnosis in the absence of neuritic symptoms and of an alcoholic history at the time. In the post-hallucinatory condition it was doubtful whether patient would regain his retentive power, or pass into a condition of Korsakoff's psychosis. Such complete recovery is extremely rare.

The second case reported is typical of Korsakoff's psychosis; the author discusses the symptoms in detail.

C. MACFIE CAMPBELL.

**POLYNEURITIS CEREBRALIS MENIERIFORMIS.** BERGER, (364) *Neurolog. Centralb.*, Sept. 16, 1905, p. 844.

UNDER this title, von Frankl-Hochwart described a symptom-complex apparently of an acutely infective nature, consisting in

paralyses of various cranial nerves, on one side only. As the name implies, symptoms akin to those of Menière's disease are present in the shape of tinnitus, giddiness, diminution of auditory acuity, vomiting, etc. A succinct resumé is given of the few cases hitherto recognised and reported.

The author's own case was a man 55 years old, with some indications of general arterio-sclerosis. On the 28th December 1904, after a prolonged exposure to cold air, he began to suffer from malaise and was slightly fevered. On the morning of the next day intense giddiness occurred whenever he tried to get on his feet, and he thought the right side of his face was stiff. Examination on the following day revealed a slight swelling of the tissues round the right ear and over the parotid, together with a crop of herpetic vesicles on the pinna and in the external auditory meatus. There was complete peripheral facial palsy on the right side, while both skin and mucous membranes on that side were hyperæsthetic. The point of exit of the trigeminal was excessively tender. Taste was blunted on the right anterior part of the tongue, and hearing too was diminished. The giddiness was particularly severe. Very considerable improvement took place in the course of the succeeding two months, but the condition did not clear up entirely. It was obvious that an acute toxic process of some kind or other had caused a polyneuritis of several nerves, which in the majority of the recorded cases have been the eighth, seventh, and sensory fifth on one side. S. A. K. WILSON.

**PARTIAL MYOTONIA OCCURRING IN A CASE OF OCCUPATION-NEUROSIS AND PARALYSIS.** (Ueber partielle Myotonie unter dem Bilde einer Beschäftigungs-neurose und -lähmung.) HANS CURSCHMANN, *Berlin. klin. Wochenschr.*, Sept. 11, 1905, p. 1175.

CURSCHMANN records the following case. The patient was a brush-maker, with no previous personal or family history of nervous trouble; his occupation involved great strain, specially of the extensors of the right wrist. At age 17, gradually increasing weakness appeared in the radial extensors of the right wrist, and later in the extensors and abductors of the right thumb. Some time after this paresis was evident, the adductor and opponens muscles of the right thumb, and later those of the left thumb, began to suffer from a condition of cramp, induced by any movement of these muscles, so that, after contraction, as in clasping a tool, they could not be relaxed for some time; this cramp never occurred spontaneously, and was never accompanied by pain or paræsthesia.

The same "myotonic" condition appeared soon in the right flexor carpi ulnaris. One and a half years after onset the following two main facts were noted: (1) Complete paralysis and atrophy of the radial extensors of the right wrist and of the extensors and abductors of the right thumb, slight paresis of the ulnar extensor of wrist and of supinator longus on right side, and of abductor pollicis and perhaps of extensores pollicis on the left side. (2) A typical myotonic condition, with characteristic reactions, in the right opponens pollicis and probably also in the right adductor pollicis, in the right flexor carpi ulnaris and in the left opponens pollicis. No trace of paresis, myotonia, or other abnormality was found in other muscles, except that the tongue showed mechanical reactions characteristic of myotonia. The electrical excitability of all nerves, motor and sensory, was normal, but the facial nerves were markedly over-excitabile on mechanical stimulation (Chvostek's sign). All tendon reflexes were abolished.

Curschmann believes that this case is unique in as far as it shows a true antagonism between the paresis and the myotonic condition, the paresis occurring first, as result of "professional" overstrain of muscle, in a subject predisposed to myotonia (*cf.* the tongue condition), and the myotonia manifesting itself, secondarily, in the antagonistic flexors which are in a hypertonic state and are, moreover, muscles showing special tendency to suffer from the myotonic condition in typical cases. The strict localisation of the myotonia in this case is certainly very remarkable. The author can find no case on record—with the possible exception of a case reported by Jolly—in which "professional" muscular overstrain has been thought to have had any effect, direct or indirect, in exciting a myotonic condition; he suggests that it would be well to search for myotonic phenomena in spastic forms of occupation-neuroses.

Not a few cases of Thomsen's disease, with muscular atrophy, are to be found in literature; in some cases the distribution of the atrophy is like that found in idiopathic muscular atrophy of the juvenile type, in other cases like that of the Duchenne-Aran form of amyotrophy, and in other cases like a neuritic atrophy. Curschmann's case differs from practically all these cases in the distribution of the muscular atrophy, in the escape of the facial muscles, and in the absence of myotonic reactions in the atrophied muscles.

It is not always easy to differentiate between tetany and myotonia, more especially as the spasms of tetany may occur only on movement, disappearing during rest. Cases have been described of tetany and myotonia combined, and the presence of Chvostek's sign in Curschmann's case, as in several other recorded cases of myotonia with amyotrophy, is bound to rouse the suspicion of such

a combination here. Every other sign of tetany, however, was absent, and this is regarded by the author as certain proof against the existence of any tetany element.

Attention is drawn to the remarkable absence of tendon reflexes, even those of the legs, which showed no paresis or myotonia or even hypotonus. This loss of tendon reflexes has been noted in a considerable proportion of cases of myotonia with muscular atrophy, and has to be reckoned as a symptom, not as a complication.

Remarks are made on the electrical reactions of the myotonic muscles; these agreed largely with Erb's classical description, but not entirely. Thus no absolute increase in the direct excitability of the muscles either to faradism or galvanism was found, and there was no slowness in the rise of the contraction wave.

A. W. MACKINTOSH.

**ON THE RESULTS OF SMALL LESIONS IN THE REGION OF  
(366) THE MOTOR ARM CENTRE, INCLUDING A CONTRI-  
BUTION ON CYSTICERCUS CEREBRI. (Über Folgezustände  
kleinster Läsionen im Bereiche des motorischen Armsentrums  
nebst einem Beitrag zu Cysticercosis cerebri.)** Dr OSKAR  
FISCHER, *Monatsschr. f. Psychiatrie u. Neurologie*, Bd. 18, H. 2.

As opposed to Bonhoeffer's theory that any partial lesion in the motor centre of a limb will cause a greater paralysis in the distal and more highly functioning parts of the extremity, there is Stark's case in which, with a cystic tumour in the arm centre, the corresponding shoulder showed the most limitation of movement. Other similar cases have been reported by Pick, Gros, Stimson, Lapine, and Fox, and the following case also seems to contradict the truth of this theory:—

The patient was the subject of numerous Jacksonian attacks, starting in the little finger of the left hand, and spreading up the arm to the face. He had numerous small subcutaneous nodules, one of which on removal proved to be a cysticercus cyst. At operation a cyst over the arm centre in right hemisphere was removed. Faradic stimulation in front of the site of the cyst caused movements of the forearm muscles; behind, caused tetanic abduction of the thumb. As this induced a general epileptic fit, the site of the lesion could not be more accurately localised. After the operation there was complete paralysis and anæsthesia of the area, but this improved slowly until only a difficulty in moving the fourth and fifth fingers remained, with some anæsthesia over these fingers.

Seven months later the fits recurred; at first, as usual, starting in the left hand, then later they started with paræsthesiæ in right foot and hand. This led to a second operation on the left hemisphere, but nothing pathological was found. Four years later he again came under observation, and then showed some awkwardness and difficulty in moving the fourth and fifth fingers of the left hand, in closing hand and in flexing and extending the wrist, with some relative anæsthesia and analgesia of the third, fourth, and fifth fingers, and some loss of sense of position.

After a genuine attack, in which head, eyes, and body turned to the left, with twitching of both upper extremities and clonic spasm of jaws, with biting of tongue, the patient developed a hysterical hemianæsthesia, and the attempt to influence this by means of Faradic current produced a series of hysterical fits, followed by complete return of sensibility, except in the two ulnar fingers, with perfect sense of position and stereognostic sense in all fingers.

The author considers that the disturbance of sensibility in the fourth and fifth fingers was purely a hysterical manifestation; the paresis of the fingers was undoubtedly organic in origin, probably softening due to vascular occlusion following operation. He concludes (1) that the patient had a centre for isolated movements of the two ulnar fingers; and (2) that this centre is situated, as Krause has already found, between the motor centre of the thumb and that for movement of the wrist.

COLIN K. RUSSEL.

**PSEUDOMELIA PARÆSTHETICA AS A SIGN OF DISEASE IN  
(367) THE NEIGHBOURHOOD OF THE LENTICULAR NUCLEUS.**

(*Pseudomelia paraesthetica als Symptom einer Cerebralaffectio-  
im Gebiete des Linsenkernes.*) BECHTEREW, *Neurolog. Centralb.*,  
Sept. 1, 1905, p. 786.

THE author proposes to apply the involved and enigmatical term "pseudomelia paræsthetica" to subjective sensations of movement in completely paralysed limbs. In a case of cerebral softening in the neighbourhood of the right lenticular nucleus, a prominent and permanent symptom was the false impression the patient had of frequent movements in his paralysed left arm. The fact that he could convince himself of the absolute immobility of his arm by looking at it made no difference in the phenomenon. The obvious analogy is drawn with what commonly obtains in the case of amputated limbs. To say that the condition depends on alteration in the conduction of muscle sense is very inadequate.

S. A. K. WILSON.



**HEMIATAXIA IN A CASE OF TRAUMATIC HEMIPLEGIA.**

(368) **RECOVERY AFTER TREPHINING.** GAUSSEL AND MASS-  
BAU, *Arch. gén. de méd.*, Oct. 3, 1905, p. 2518.

THE writers give full details of the case of a young mechanic in whom paresis on the right side and mental impairment followed a severe blow on the left side of the head. He remained unconscious for ten days, after which he gradually aroused himself, though marked paresis remained in the right arm and leg, together with great inco-ordination, affecting especially the right lower limb on walking and performing other voluntary movements, so that the man was quite unable to work. No impairment of sensibility and practically no involvement of the reflex functions. Three months after the accident the patient was trephined, when a depressed fracture, with splintering of the inner table and formation of a callus 12 mm. high, was found near the left fronto-parietal suture and removed. The meninges were unaffected. Shortly after the operation, marked improvement was seen and continued to increase. The mental recovery was complete, and, though slight weakness persisted in the right arm and leg together with slight ataxia in the latter, the patient was able to resume his work.

JOHN D. COMBIE.

**THE INFLUENCE OF CEREBRAL DISEASE ON MENSTRU-**

(369) **ATION.** (Über die Beeinflussung der Menstruation durch  
cerebrale Herderkrankungen.) MÜLLER, *Neurolog. Centralb.*,  
Sept. 1, 1905, p. 790.

WITH a view to substantiating and elucidating the relation between amenorrhœa and cerebral tumours in certain positions, the symptom was carefully sought for in a series of cases, of which five are reported in this paper. Three were cerebellar tumours: one was in the posterior fossa, the other was not accurately diagnosed.

In each amenorrhœa was an early and a persistent sign. There are obviously three possibilities. The idea that the brain lesion is secondary to the amenorrhœa is not to be entertained. In the next place it is conceivable that the two conditions might own a common cause. This theory is highly improbable, seeing that the combination occurs so seldom, though each is very far from being rare.

It is much more likely that the amenorrhœa is directly dependent on the cerebral lesion.

Psychical influence on the menstrual function is of course a familiar fact. In acromegaly, ovarian and mammary atrophy has been described, associated with the symptom under discussion. A similar complex has been noted by Axenfeld in basal tumours. It would appear that tumours of the hypophysis and of the adjoining areas, as well as others in different situations that are attended by early and well-marked hydrocephalus and troubles of vision, are especially liable to announce themselves by one amenorrhœa. Whether the hydrocephalus mechanically disturbs pituitary function, which in its turn has a prejudicial action on the ovaries, either by the blood or directly through the nervous system, remains to be established.

S. A. K. WILSON.

**CHRONIC PARALYSIS OF THE INTERCOSTAL MUSCLES AS (370) A PRIMARY CAUSE OF DROPSY.** GOLDWIN W. HOWLAND, *Lancet*, Oct. 7, 1905, p. 1035.

A LAD sustained a blow on the neck producing a dislocation of one of the cervical vertebræ. Myelitis followed, affecting the 5th, 6th, 7th, and 8th cervical regions of the cord. Sensation returned, but atrophy of the muscle groups represented in this region occurred, accompanied by a spastic paralysis of the legs.

The intercostal and abdominal muscles being paralysed, respiration had been entirely diaphragmatic for four years, resulting in diminished expansion of the upper lobes of both lungs, with consequent obstruction to the venous circulation and to the right side of the heart.

During the last few months, dropsy appeared in the legs, followed at a later date by œdema of the neck, face, eyelids, uvula, and larynx.

Paralysis of the intercostal muscles is probably the rarest of the respiratory agencies which tend to produce dropsy.

H. DE M. ALEXANDER.

**RETRO-OCULAR NEURITIS.** R. MARCUS GUNN, *Ophthalmic Review*, (371) Oct. 1905, p. 285.

THIS important paper was read at the Oxford meeting of the British Medical Association. It begins by discussing the diagnosis of neuritis where the papilla is not involved, and in the differentiation of retro-ocular neuritis from an ordinary papillitis, when the former is so far forward as to involve the papilla. The diagnosis rests on definite clinical symptoms. "Rapid failure of vision, often in one eye only, particularly affecting the macular area, and commonly accompanied by pain and tenderness in the orbit . . .

impaired pupil reaction to light, frequent absence of decided early ophthalmoscopic changes and a tendency to recovery. Occasional anomalous forms exist." Does a perineuritis exist? There may be difficulty in some case in diagnosing these cases from macular affections. The pupil reactions usually suffice to differentiate the cases from functional amblyopias. The failure of vision may vary from only slight dimness with only relative central colour scotoma up to temporary complete blindness. The visual acuity is diminished by exposure to bright light, and often things are seen as through a heat haze (possibly due to impaired insulation in the nerve fibres). Light-sense is decidedly affected, and even in the lightest cases there is nearly always a complete or a relative colour scotoma for red and green at the fixation point.

The central portion of the visual field is always most affected. Why the macular bundle should be thus picked out is uncertain. Three explanations have been suggested: (1) That the principal lymph current flows in the neighbourhood of that bundle. (2) Silcock's suggestion that for the greater part of the nerve the central fibres are entirely dependent on peripheral vessels for their supply. (3) The macular fibres being most highly specialised physiologically, suffer most rapidly from any degenerative process.

The visual failure is rapid, but not sudden. The prognosis is on the whole good.

Pain is present in a large percentage of cases, usually preceding loss of vision. It was sufficiently severe to be specially noted in 120 cases out of 360 collected for this paper.

Early changes of the disc of an inflammatory nature occurred in 101 out of 350 cases. Pallor of the disc of varying degree is probably almost universal as a sequel sooner or later following the neuritis.

The etiology is varied, and in some cases obscure. They can be roughly divided into two large classes:—

*A.* Inflammation communicated to the nerve from neighbouring structures, as orbital cellulitis, orbital periostitis. Disease of the sphenoidal sinus, rheumatic inflammations of the sheath of the nerve in the optic canal, and inflammation at the scleral opening, analogous to the episcleritis of the anterior segment of the eye. One hundred and eleven cases belonged to this class.

*B.* Inflammations or other rapid changes occurring initially in the optic nerve. This class can be sub-divided into two subclasses: (*a*) local manifestations of a general disease, such as syphilis or tuberculosis; and (*b*) primary retro-ocular neuritis, an ill-defined group, where the inflammation cannot be shown to be secondary to inflammation in surrounding structures. Two hundred and twenty-three cases of this kind included 51 due to insular

sclerosis. Of the remaining 172, no definite causation was found for 55. Influenza accounted for 27; gout for 22; and various other causes, mainly toxic, were found in the remaining 68.

The pathological nature of the nerve affection in insular sclerosis is discussed, and in the concluding paragraph of a most valuable paper the sex and age distribution of the 360 cases is given.

LESLIE PATON.

**A CONTRIBUTION TO THE KNOWLEDGE OF THE GENESIS OF (372) UNILATERAL OPTIC NEURITIS.** (*Ein Beitrag zur Kenntniss der Genese einseitiger Stauungs-papille.*) SCHULTZ-ZEHDEN, *Klin. Monatsbl. f. Augenheilk.*, Aug. 1905, p. 129.

SCHULTZ-ZEHDEN describes the case of a patient 45 years of age. During life the diagnosis lay between cerebro-spinal syphilis and cerebral tumour. The post-mortem examination revealed a large cholesteatoma on the right side of the base of the brain. The man suffered from mental weakness and dementia, and he had fits, with loss of consciousness. His reflexes were normal and there was no loss of common sensation. His musculature was good.

Ophthalmoscopic examination of the right eye, which was completely amaurotic, with fixed pupil, showed a snow-white disc with sharp edges, small vessels, and no evidences of any swelling—a picture of simple atrophy.

The left eye (visual acuity = fingers at four metres) showed an optic neuritis with four dioptries of swelling, colour grey, with a reddish shimmer. Vessels curved over edge and occasionally buried. Veins dilated, arteries narrow. A condition of optic neuritis passing into atrophy.

During life, Schultz-Zehden had given, as a possible explanation of the appearances, a tumour pressing on the right optic nerve and causing a descending atrophy, while setting up a normal optic neuritis on the left side from rise of intra-cranial pressure. At the post-mortem examination a large cholesteatoma was found on the right side of the base of the brain, which compressed, amongst other things, the right optic nerve from the chiasma forwards to the optic foramen.

LESLIE PATON.

**A CLINICAL CONTRIBUTION ON DISEASE OF THE OPTIC (373) NERVE RESULTING FROM ATHEROMA.** (*Beitrag zur Klinik der Sehnervenerkrankungen infolge von Gefässatheromatose.*) Dr B. STÖLTING, *Klin. Monatsbl. f. Augenheilk.*, August 1905, p. 113.

In this paper Dr Stölting describes in full detail four cases in which the changes in the optic nerve and the various visual

symptoms were, he considers, due to atheroma affecting the internal carotid, the ophthalmic or the retinal arteries. In the introductory part he cites a few cases which have been previously described, and discusses the manner in which disease in these various arteries may affect the optic nerve.

The first of Dr Stölting's cases was in a lady, 48 years old when first seen. She complained of diplopia, which had lasted about 18 months, and which was due to paresis of both external recti. Her vision (with myopic correction) was at this time good, and there were no ophthalmoscopic changes, except a myopic crescent. A year later (July 1887) the separation of the images had increased. In the autumn of 1895, nine years after the first visit, she came complaining of commencing haze and difficulty in seeing in the twilight. Visual acuity, R.E. =  $\frac{6}{18}$ , L.E. =  $\frac{6}{16}$ . Both discs showed neuritis. Left more swollen than the right, and both showed a small hæmorrhage. The condition of the eyes got rapidly worse, with diminution of vision and contracting fields. Her urine, heart, and lungs were normal. The condition was thought to be due to cerebral tumour. A post-mortem examination revealed no signs of tumour, but an extensive sclerosis of the cerebral vessels. Unfortunately the relationship of the vessels to the optic nerve was not noted.

The second patient was a man who also complained of diplopia. He had a three years' history of epileptic fits, with occasional giddiness and paresis of left leg, and occasional paræsthesias of the left hand. The diplopia was due to double external rectus paresis.

His vision was—R.E. =  $\frac{4}{18}$ , L.E. =  $\frac{4}{8}$ , with -1 D. sph. in E.E. This loss was due to optic neuritis, more marked on the right side than on the left. The visual fields were full. The optic neuritis kept on in a chronic form. Later in the same year (1889) an attack of apoplexy paralysed the left extremities. After this attack he was examined, and his visual acuity was found to be—R.E. =  $\frac{2}{30}$ , L.E. =  $\frac{4}{24}$ . Visual fields both narrowed, especially in the lower nasal segment. Optic neuritis more advanced, with swelling of 1.1.3 mm. At this time the symptoms pointed to cerebral tumour. The symptoms rapidly became worse. By March 1901 the vision was reduced to counting fingers at one metre, and optic atrophy had set in. The patient died shortly after.

The third and fourth cases give somewhat similar histories, but in addition to optic nerve affection with diminution of sight and contraction of the visual fields, the retinal arteries in these cases showed definite evidences of arterio-sclerosis.

We regret that space does not permit of our giving a fuller abstract of Dr Stölting's cases. He seems to be quite ignorant of

the work that has been done in England on this subject, and some of his descriptions might have been translated directly from Gunn's descriptions of similar cases.

LESLIE PATON.

**ACROMEGALY AND BLINDNESS.** (*Acromégalie et Cécité.*) M. A. (374) CANGE, *Arch. gén. de méd.*, Oct. 10, 1905, p. 2575.

A CLINICAL lecture upon a case of acromegaly occurring in a coachman aged 25 years; weight, 90 kilos.; height, 1·7 metres. The family history was negative. Patient suffered from malaria for 18 months when 5 years of age, was treated with quinine, and has had no return of the disease; no history of venereal trouble, and patient quite healthy until three years ago, when, at the age of 22, he became irritable and melancholic and took to drink, and also began to get much stouter. The author then describes the case fully, calling attention to the presence of enlargement of the face, especially of the lower jaw; thickening and eversion of both lips; enormous enlargement of the tongue, and changes in the mucous membrane of the isthmus of the fauces and of the pharynx. He also describes, as symptoms of somewhat less importance, the occurrence of headache, somnolence, great muscular weakness, polyuria (no albumin or sugar present), deficiency of urea, etc. He then specially emphasises the alterations in the hands and feet, and the occurrence of visual complications. The power of vision began to diminish about a year before admission, at first gradually, then rapidly, until total blindness supervened in three months. This was unaccompanied by any inflammatory symptoms or pain, and was described by the patient himself as being like a veil which seemed gradually to increase in thickness. On ophthalmoscopic examination, atrophy of both optic nerves was found, the discs being very white and distinct in outline, and the vessels being apparently unaltered in calibre. These changes were more marked in the right eye, on which side there was also present a divergent amblyopic strabismus; the pupillary reaction was sluggish, and there was complete blindness. On the left side a light placed at a distance of one metre from the eye gave rise to a sensation of diffuse illumination only, there being no apparent difference in the two halves of the field of vision.

The writer then discusses the differential diagnosis of the case with regard to Paget's disease, chronic hypertrophic pulmonary osteo-arthritis, and leontiasis ossea of Virchow. Visual complications are observed in 73 per cent. of cases of acromegaly (Hertel), and may or may not be accompanied by any visible

changes in the fundus. Most frequently ophthalmoscopic examination reveals the presence of optic atrophy, of which there are three possible explanations:—

1. Following upon a neuritis of the optic nerve, toxic in origin.
2. Swelling and œdema of the papilla from compression of the cavernous sinus, or from other causes, as in cerebral tumour generally.
3. Simple primary atrophy from pressure upon the optic commissure and nerves by a pituitary tumour or enlarged sphenoidal sinus, or from narrowing of the optic foramina (Broca, Duchesneau).

In the case described, the optic atrophy is probably due to the third of these causes, namely, simple primary atrophy from pressure, and the prognosis is a grave one on account of the early onset and rapid advance of the visual complications. Treatment with strychnine, electricity, pituitary and, if failing, thyroid extract, the probable success of which, however, is somewhat doubtful, will be tried.

W. E. CARNEGIE DICKSON.

**THE NEGATIVE VALUE OF KERNIG'S SIGN.** WILLSON, *Amer. (375) Journ. Med. Sciences*, Aug. 1905, p. 306.

WILLSON examined a series of 73 adults and 47 children, who were free from symptoms of meningeal involvement. The sign was considered to be positive only if one or other leg, when raised by the heel, without an effort that could properly be termed force, presented a distinct flexion angle, varying from 120° to 110° at the knee, when the thigh was at right angles to the trunk. Thirty-two of these cases, *i.e.* 26·8 per cent., presented the sign in the absence, clinically speaking, of meningitis. One must conclude that it is by no means a positive indication of either meningeal or brain or cord involvement, but that when present in association with other signs of meningitis it may be looked upon as confirmatory to a high degree. Undoubtedly in a majority of cases it persists far into the convalescence, and is usually one of the last to disappear. In certain cases it probably remains as a permanent indication of a former undetermined lesion. S. A. K. WILSON.

## PSYCHIATRY.

**PROBLEMS OF CLINICAL PSYCHIATRY.** (*Fragestellungen der (376) klinischen Psychiatrie.*) E. KRAEPELIN, *Centralbl. f. Nervenh. u. Psych.*, Aug 1, 1905.

In this lecture Kraepelin passes in review briefly those questions in psychiatry which at the present moment press most urgently

for a solution. He first mentions amentia. In this group are frequently included cases which, by their later course, prove to be examples of dementia præcox, or of manic-depressive insanity. With regard to the remaining cases, we have as yet no sufficient means of distinguishing them from those with different outcome. Kraepelin differentiates a small group with severe somatic disorders and good prognosis: he cannot assert that similar cases may not also pass into dementia, but if they do, he assumes that the terminal condition must present its own characteristic features, different from those seen in dementia præcox. Sufficient attention has not been given to the mental impairment following acute infections.

Alcoholic psychoses present many interesting questions, both of ætiology and symptomatology; the correct appreciation of deteriorated cases with a paranoid trend is especially difficult; their relation to the deterioration in dementia præcox is not known.

With regard to dementia præcox, Kraepelin insists on the provisional nature of the group. In view of the transitions between the hebephrenic and the catatonic complexes, it was better to recognise them as forming one big group with various forms.

The name has a historic justification, but is misleading, and must not be taken with its original connotation, for catatonia may have its onset in advanced years, and one cannot deny the possibility of complete recovery in cases of dementia præcox. Kraepelin would welcome a more appropriate name. All his efforts to satisfactorily differentiate the huge group have been unsuccessful. In the absence of much light from the side of pathological anatomy, investigation must be directed to the terminal conditions. The various groups which may be here separated, depending upon some difference in the morbid process or in its localisation, may enable us retrospectively to differentiate the earlier stages. The material for such an investigation is to be found in the huge nucleus of deteriorated cases in insane asylums. Kraepelin is much influenced by the analogy of dementia paralytica with its definite progressive morbid changes, leading to certain terminal conditions varying with the intensity and localisation of the process.

In discussing the origin of his large group of dementia præcox, he justifies the union of the hebephrenic and catatonic forms, but does not refer to the inclusion of the paranoid form. When he refers later to paranoia, he is less inclined than in his text-book to sweep into his dementia præcox delusional conditions such as Magnan's *délire chronique à évolution systématique*.

There are many questions as to general paralysis demanding an answer. Why is it in some cases stationary? Must it have a



fatal outcome? Post-mortem examination of the stationary cases is of the greatest importance.

The mental disorders of advanced life await more accurate study. Catatonia and manic-depressive insanity may first manifest themselves at this age. It is difficult to establish the clinical relations of a depression, with recurrence, especially if deterioration ensue. The depressions in the involution period have not been satisfactorily differentiated.

Manic-depressive insanity furnishes still other problems: the variations in its forms, especially the mixed forms, are only imperfectly understood. The prognostic importance of the latter and their relation to preceding attacks require investigation. What about the manic cases which simmer down into a "chronic mania"? What is the relation of chronic constitutional depressions or excitements to this group with its well-defined attacks.

Epilepsy needs further differentiation. Kraepelin rejects a transitional hystero-epilepsy, but admits definite psychogenic episodes in epileptics. Hysteria does not limit itself to superficial and fleeting disorders; psychogenic mental disorders may persist for years.

In conclusion, Kraepelin points to the vast amount of useful material in the great insane institutions; he calls for a large and accurate series of observations of patients covering years and decades, and hopes by the statistical accumulation of trustworthy material to determine more accurately various clinical groups and enable a more definite prognosis to be given on the individual case. It is for a psychiatric clinic to stimulate and direct investigation and elaborate more accurate methods, but the co-operation of those who work in the asylums is necessary to give a sound basis to a clinical psychiatry.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO THE KNOWLEDGE OF KORSAKOFF'S  
(377) PSYCHOSIS, WITH SPECIAL REGARD TO ITS PATHO-  
LOGICAL ANATOMY. A FURTHER CASE. (Beitrag zur  
Lehre von der Korsakoffschen Psychose mit besonderer  
Berücksichtigung der pathologischen Anatomie. Ein weiterer  
Fall.) G. WEHRUNG (of Strassburg), *Arch. f. Psych.*, Bd. 39,  
H. 2.**

WEHRUNG first gives a short historical sketch and then passes to the discussion of the ætiology of Korsakoff's psychosis. Chronic poisoning by alcohol is by far the most frequent cause, and according to the author it acts through the heaping up in the system of substances analogous to antitoxins: the presence of such sub-

stances in the blood would account for the onset of delirium tremens in a sober period when there is no alcohol in the circulating fluids. Alcoholism is, however, only a predisposing cause, and does not *per se* give rise to the psychosis; the precipitating cause may be another intoxication—trauma, fever, an apoplectic or epileptic attack, etc. Alcoholism is not the only predisposing cause: chronic metallic poisoning, trauma with neurotic sequels, fevers with their toxins and antitoxins, and tumours leading to cachexia, are other causes. Where there seems but one cause, the auto-intoxication due to gastro-intestinal disorder is probably also an ætiological factor, *e.g.* in alcoholism and typhoid fever.

The author, therefore, considers that Korsakoff's psychosis is caused by a chronic toxæmia, and is precipitated by various debilitating circumstances. Those who do not accept this ætiology adduce as their reason the occurrence of the psychosis after trauma, in brain tumour, and as a senile change. Wehrung could find no record of a case arising on the basis of trauma alone, which he admits merely as a predisposing or precipitating factor. In brain tumour one cannot exclude toxic action. He denies the occurrence of the psychosis as a senile change; there one may meet a psychosis with the same memory defect as the main feature, but the progressive narrowing of the intelligence, the want of mental alertness, the selfish interests of the senile, with the various episodes and different development constitute an essentially different psychosis.

The discussion of the symptomatology is introduced by a quotation of Korsakoff's original statement in 1890. Korsakoff described a psychosis beginning with a delirium, which passed either into a chronic delirium described as stuporous dementia, or into an apathetic confusion: in addition to the mental symptoms, physical symptoms were always present, the most important being multiple neuritis. Korsakoff soon removed the stuporous dementia from the picture of the psychosis, leaving the apathetic confusion, in which the most striking feature was marked memory defect for events since the onset of the psychosis. Later authors defined more clearly the psychosis, recognised that it may arise without an initial delirium, and that it need not be accompanied by polyneuritis. The latter, however, is almost constant, and was frequently demonstrated on microscopic examination when the clinical examination gave negative results. The psychosis is characterised by poor retention, disorientation in place and time, and fabrications: the apathy and the confusion are secondary to the memory defect. The author calls attention to the fact that—(1) a total general amnesia never occurs; (2) the period of amnesia has no sharply defined limit; and (3) in the amnesic period, isolated remnants are found. The amnesia frequently ex-

tends further back than the onset of the psychosis. The nature of fabrications is discussed in detail: patient not only has falsifications of memory, owing to which actual events are hopelessly jumbled and described in absolutely false sequence and connection, leading to rather fantastic stories, but he may have also hallucinations of memory, and recall confidently events which never happened. This recalling of imaginary events arises in conversation when there occurs to the patient some picture which he proceeds to identify as an actual past experience. To the latter of the two memory phenomena Wehrung would limit the term "pseudo-reminiscences"; while he uses "confabulations" for the fabrications which a patient uses to bridge over awkward gaps in his memory.

With regard to the diagnosis, the author points out the points of difference between this and the similar senile psychosis: he makes rather light of the difficulty of diagnosing it from some cases of general paralysis. A table is given showing briefly the pathological findings in 34 cases of Korsakoff's psychosis recorded in the literature. Neuritis was found in all cases where it was looked for: several authors recorded diminution of the tangential fibres in the cortex; the cell-changes found were not distinctive.

In conclusion, the author records a personal case, the patient being a tubercular alcoholic with classical symptoms; microscopical examination showed parenchymatous neuritis, degeneration of Goll's tract, and of the posterior and anterior roots, with some change in the anterior cornual cells in the lumbar region. In the cortex there was a considerable diminution of the tangential fibres.

C. MACFIE CAMPBELL.

### TREATMENT.

**THE DIETARY OF EPILEPTICS.** (*Le régime alimentaire des (378) épileptiques.*) JULES and ROGER VOISIN, *La Presse Médicale*, Sept. 2, 1905, p. 555.

PROLONGED experimentation has convinced the authors of the therapeutic ineffectiveness of a purely milk diet. Further, any value found in the combination of a milk-vegetarian diet with abstention from salt is to be attributed exclusively to the reinforcement of the action of the bromides by the withholding of the sodium compound, whatever be the explanation of the fact.

Of course there is no reason *a priori* why an animal diet any more than a vegetable diet should produce signs of digestive auto-intoxication, and a series of observations were carried out on ten epileptic children belonging to the Salpêtrière, in order to provide

experimental evidence on this point. During a month their nourishment consisted solely of vegetable food, in which, moreover, the nitrogenous elements were reduced to a minimum. Sugar was added to supplement the diminution of calories consequent on the suppression of nitrogen. All else—exercises, wine, salt, potassium, bromide, etc.—remained unchanged. The results were absolutely negative.

The general conclusion must then be that the prescription of a milk-vegetarian régime in epilepsy is based on erroneous views of auto-intoxication and on inaccurate knowledge of digestive phenomena. Its adoption is not only unnecessary but useless, whereas the maintenance of general hygiene and the avoidance of digestive troubles are all that is called for.

S. A. K. WILSON.

**ON THE MECHANICAL TREATMENT OF NEURALGIA.** SACHS,  
(379) *Wien. med. Wochenschr.*, Sept. 2, 1905, p. 1750.

THIS is a commentary upon papers which had appeared in the same publication, No. 23 of 1905, and Nos. 1-4 of 1904, upon the value of massage in neuralgia. The writer records some of his satisfactory experiences in the treatment of sciatica by massage, combined with electricity and baths.

JOHN D. COMRIE.

**THE QUESTION OF THE PERMANENCE OF THE RESULTS OF  
(380) OPERATIVE TREATMENT OF EXOPHTHALMIC GOITRE**  
(Ueber Dauererfolge nach operativer Behandlung des Morbus Basedowii.) FRIEDHEIM, *Arch. für klin. Chir.*, Bd. 77, Heft 4, p. 917.

THE author gives details of the twenty cases of exophthalmic goitre from which he has removed portions of the thyroid gland. Of these cases, fourteen are completely cured, and intervals of from four to fifteen and a half years have elapsed since operation. Marked improvement occurred in two patients; while in three cases there was considerable improvement, but symptoms remained or returned sufficient to justify the recommendation of a further operation. One patient died suddenly on the tenth day.

While admitting that Basedow's disease is due to a hypersecretion from the thyroid gland, some object to operative treatment on the ground that the surgeon can only remove part of the gland, and that therefore the production of poisonous material continues. The answer is that enough gland tissue must be removed to prevent this, while sufficient must be left to avoid

myxœdematous symptoms. The determination of the quantity to be excised constitutes the most difficult and important point in the operative treatment. Kocher's advice is worthy of consideration—rather to remove smaller pieces on several occasions than to risk too extensive an excision. As alarming symptoms may immediately follow operation, apparently from absorption of the poison-laden blood, thorough arrest of hæmorrhage is essential, a preliminary ligature of afferent vessels being performed if necessary.

Statistics from various hospitals give 69 per cent. as the number cured by operation, and the mortality as only 7 per cent. As the mortality of the disease is 12 per cent., and permanent cure by non-operative means is practically unknown, "there undoubtedly exists the right, indeed the duty, of recommending operation, at least in such cases as defy medicinal treatment."

W. J. STUART.

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# **Review**

of

## **Neurology and Psychiatry**

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### **Original Articles**

#### **A NOTE CONCERNING MESOGLIA CELLS.**

By JOHN TURNER, M.B.

IN 1900, Ford Robertson gave the name mesoglia to little branched cells, very distinctly shown in the brain by his platinum method, to differentiate them from the ordinary neuroglia cells, which he looks upon as of an entirely different nature. The name mesoglia denotes their origin from mesoblastic tissue.

The object of this note is to point out that cells having all the characters of those described by Ford Robertson as mesoglia, occupy the peri-vascular and peri-cellular spaces of the cortex, and lie in close proximity to the nerve cells, and that their branches ramify extensively and form the closely fitting trellis-like Golgi net enveloping the nerve cell and its processes.

With my methylene blue and peroxide of hydrogen process, under certain conditions not yet accurately determined, these tiny elements stain out jet black, whilst the pyramidal nerve cells stain a pale turquoise blue, and the matrix a very pale grey with a tinge of red. Under these circumstances it is possible to estimate accurately the precise relationship of these mesoglia cells to the other elements.

I will describe them as they appear with my method, and it will be found that the description tallies closely with that given by Ford Robertson in his Text-book of Pathology.

The cells are very irregular in shape, and smaller than any nerve cell in the cerebral cortex with the exception perhaps of

some of the so-called granules; they rarely exceed 7 by 5  $\mu$  and are often smaller. The cytoplasm is scanty, black, and, as Ford Robertson points out, granular; but this condition is often masked by the dense staining. The nucleus, occupying the major part of the cell body, is often paler. The branches are comparatively short, and very twisted, so that they do not lie for long in one plane; they divide and sub-divide again at short intervals dendritically, and they often turn backwards towards the parent cell quite unlike the processes of a neurone; they seem to be given off all round the cell, and are usually extremely delicate, consisting of minute dots separated a little way from each other; by this means they can often be distinguished from any extra-cellular neurofibrils which may also have stained, as these latter are quite continuous and very sharply defined. Sometimes the processes of the mesoglia cells are varicose, but the swellings are much more irregular than those on the neurofibrils.

The above description applies to mature mesoglia cells, but transitional forms are met with between these and nuclei with a mere wisp of black-stained protoplasm attached to them, and showing no processes at all; such forms are common in the white matter of the brain, and the granular layer of the cerebellum in the rat, and are met with, but in less numbers, in the human brain.

In the brain of the rat it is possible to show that the reticulum of the Golgi net arises directly from the branchings of one of the little cells lying adjacent to a nerve cell (see figs., p. 77, *Brain*, 1904); in my preparations of human brain, the Golgi net itself is not shown, but the remarkable similarity in all other respects between the Golgi net cells and those I am describing leaves very little room for doubting that they are both of like nature.

Probably these mesoglia cells are migratory, the most developed forms, those with the largest body, and most numerous and bulky branches are often found lying in the matrix of the grey, apparently some distance from a nerve cell or vessel. Furthermore, the occasional increase from morbid conditions of the number of these bodies in the peri-cellular and peri-vascular spaces points to the same conclusion.

The occurrence of apparently free nuclei in both these situations has given rise to some diversity of opinion as to their

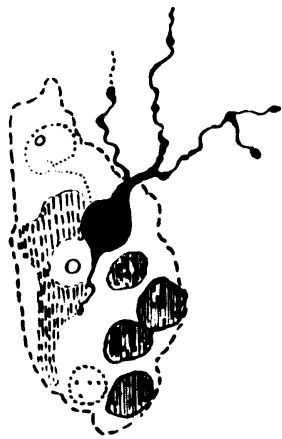


Fig 1

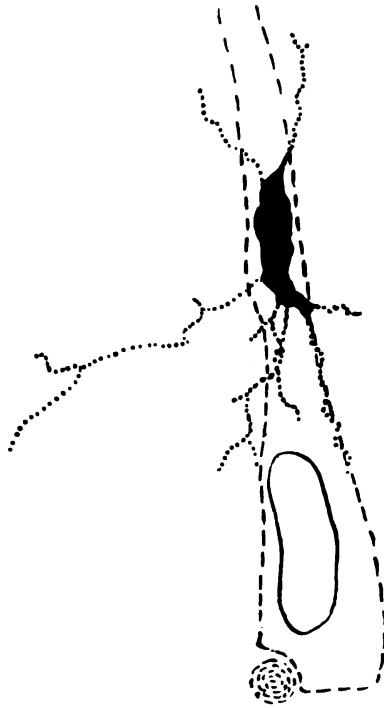


Fig 2



Fig 3



Fig 4

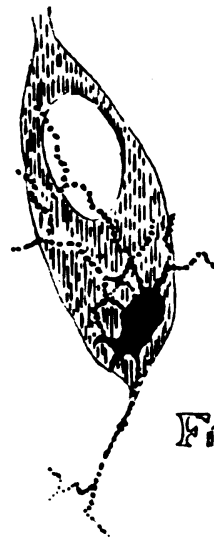


Fig 5

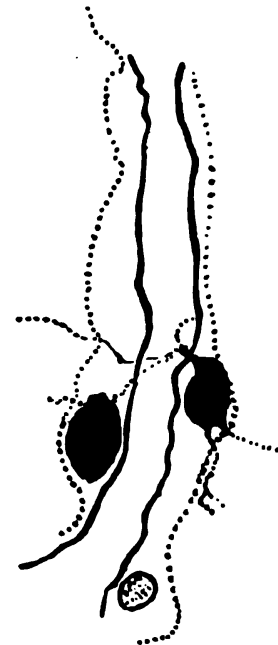


Fig 6



precise nature, especially when, as is often the case under pathological conditions, they are increased in number. My preparations very distinctly show that at all events many of these apparently free nuclei are mesoglia cells, which with the ordinary methods of staining show no, or a very slight amount of cytoplasm. The preparations show nerve cells in some instances surrounded by five or six bodies (see Figs. 1 and 3), some of which only stain a pale blue, and present the characters of free nuclei with no cytoplasm; others stain deeper and have a wisp of black stained cytoplasm attached, but no branches; others, again, present the characters of fully developed mesoglia cells. These differences of appearance are probably due in great measure to the uncertainty of the reaction which takes place between the staining fluid and the tissue elements.

#### EXPLANATION OF PLATE.

- FIG. 1.—Mesoglia cell in peri-cellular space lying close to a nerve cell. In the space, which is bounded by dotted lines, lie six other bodies, none of which show any cytoplasm. Two represented by dotted outlines stain pale blue, the other four are much darker.
- FIG. 2.—Mesoglia cell with elongated body lying alongside the apex of a pyramidal cell.
- FIG. 3.—Pyramidal cell surrounded by seven bodies; one represents a mature mesoglia cell, two have a fragment of black-stained cytoplasm but no branches, and the other four are stained pale blue and show no trace of any cytoplasm.
- FIG. 4.—Nerve cell with three mesoglia cells about it, two with branches.
- FIG. 5.—Mesoglia cell with its slender branches forming a somewhat reticulated structure over the nerve cell.
- FIG. 6.—Two mesoglia cells in a peri-vascular space, one only with branches. The edge of the space is indicated by dotted lines.

All the figures outlined with the aid of a Zeiss Camera Lucida and magnified about 800 diameters.

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**OBSERVATIONS ON BROWN-SÉQUARD'S EPILEPSY.<sup>1</sup>**

By EDWIN BRAMWELL, M.B., F.R.C.P.E., M.R.C.P.Lond.,

and

T. GRAHAM BROWN, B.Sc.

**I.—THE INCIDENCE OF BROWN-SÉQUARD'S EPILEPSY IN GUINEA-PIGS  
AS A SEQUEL TO SECTION OF THE SCIATIC NERVE.**

BROWN-SÉQUARD'S observations on the convulsive phenomena which follow certain experimental lesions are well known.<sup>2</sup> He showed that various injuries to the spinal cords of animals of different species, but especially guinea-pigs, were followed by "epilepsy, or at least a disease resembling epilepsy." For instance, he found that guinea-pigs which lived for three or four weeks after lateral hemisection of the spinal cord exhibited spontaneous attacks of convulsions, and in addition that it was possible by irritating an area of skin in the region of the face and neck on the same side as the hemisection to induce an attack. To this cutaneous area which is supplied by the infra-orbital and auriculo-temporal branches of the 5th nerve and by the upper cervical nerves, and is the only part of the body surface irritation of which is capable of exciting an attack, he gave the name of epileptogenous zone.

Later observations by Brown-Séquard and others have demonstrated that similar convulsive attacks may be produced in other ways. In the present communication we do not intend to describe the various methods by which the phenomena have been induced, but shall confine ourselves to the question of the incidence of Brown-Séquard's epilepsy as a sequel to division of the sciatic nerve.

*Previous Observations.*—It is interesting to note the

The experimental work connected with these observations was conducted in the laboratory of the Royal College of Physicians of Edinburgh, the expenses being defrayed by a grant from the Carnegie Trust. The authors take this opportunity to thank Dr Noël Paton, the superintendent of the laboratory, for his kindly advice.

<sup>1</sup> Brown-Séquard's original communication appeared in the *Transactions of the Soc. de Biologie*, ii., 1851, p. 105. Among his later contributions to the same subject we may mention his "Researches on Epilepsy: its Artificial Production in Animals," etc., published as a series of lectures in the *Boston Med. and Surg. Journ.*, Nov. 1856 to Nov. 1857.

different results obtained by observers who have performed this operation.

Thus Brown-Séquard<sup>1</sup> divided the sciatic nerve in 67 guinea-pigs, all of which subsequently exhibited the convulsive phenomena with the exception of a few instances in which the attacks were incomplete.

Obersteiner performed the same operation on 100 guinea-pigs and found that most of them showed very marked signs of epilepsy, although in a few the attacks were not fully developed. Writing in 1900, this author says<sup>2</sup> that he cannot remember whether there were any cases in which the operation was not followed by convulsive attacks, but that if there were any they were altogether exceptional.

Max Sommer in 1900 stated<sup>3</sup> that the convulsive attacks occur as a constant sequel to this operation. He based this conclusion on the fact that in every one of 40 guinea-pigs in which he divided the sciatic nerve, fits could be subsequently elicited.

These results are in marked contrast to those obtained by two other observers, Z. Gutnikow and J. P. Karplus, both of whom conducted their researches in Obersteiner's laboratory.

Gutnikow<sup>4</sup> excised a portion of the sciatic nerve in 40 guinea-pigs. In only 6 of these animals was it subsequently possible to elicit complete epileptic attacks on stimulating the epileptogenous zone; in 14 the scratching movements which constitute the initial stages of a fit were observed, while in the remaining 20 the result was negative.

The attempts made by Karplus<sup>5</sup> to induce Brown-Séquard fits by the same method were even less successful. This observer operated on 30 guinea-pigs which were kept under observation for from four to five months, but in none could a fit be produced, although in 16 the first stages of the reaction, scratching movements, were more or less distinctly seen.

Obersteiner points out that the conclusion arrived at by Max Sommer, that convulsive attacks occur as a constant sequel to division of the sciatic nerve, is an inadmissible generalisation, as

<sup>1</sup> *Archiv. de Phys.*, 1870, p. 155.

<sup>2</sup> *Neurolog. Centralbl.*, 1900, S. 498.

<sup>3</sup> *Ziegler's Beiträge zur patholog. Anatom.*, Bd. 27, 1900, S. 289.

<sup>4</sup> Quoted by Obersteiner, *Neurolog. Centralbl.*, 1900, S. 500.

<sup>5</sup> Quoted by Obersteiner, *Neurolog. Centralbl.*, 1900, S. 500.



shown by the negative observations of Gutnikow and Karplus. It is certainly a curious fact that in the experiments of 1875 a consequent epileptic state with convulsions was the rule after division of the sciatic nerve; that in 1890 it only occurred in 15 per cent. of the animals operated on; while ten years later still it did not result in 1 out of 30 guinea-pigs. That the explanation is not to be found in differences in the operative procedure seems certain, for, as Obersteiner remarks, the researches of Gutnikow and Karplus were conducted under his direct supervision.<sup>1</sup> According to Obersteiner, it would appear that the guinea-pigs of Vienna are no longer such good subjects as formerly, and that they are inferior to the animals obtained at Jena, where Sommer carried out his research.

*Personal Observations.*—Some observations of our own are of interest in this connection, since they appear to afford a possible explanation of these discordant results.

We have recently operated on 58 guinea-pigs. In each instance under ether anæsthesia the right sciatic nerve was exposed, where it lies between the trochanter of the femur and the ischial tuberosity, and a small piece (about a quarter of an inch) excised. The wound in the great majority of cases healed by first intention.

The animals were kept in roomy wire cages and well fed, two or three of about the same size occupying each cage.

In 28 of the animals at a variable period after the operation (minimum 15th day, maximum 146th day, average 36th day)<sup>2</sup> it was possible by forcibly pinching the skin of the right side of the face and neck to excite the scratching movements which initiate a typical convulsive attack. A fairly strong Faradic current applied to the epileptogenous zone was sometimes sufficient to excite the scratching, although not so effective as a

<sup>1</sup> Horsley has shown (*vide abstract of Brown Lectures*), *Lancet*, ii., 1886, p. 1212, that there is no need that the wounds should suppurate or even inflame much; even if primary union were obtained the nervous effects followed equally well.

<sup>2</sup> Brown-Séquard found that "scratching" could generally be produced between the 5th and 9th days after the operation by pinching the skin in the region of the epileptogenous zone in guinea-pigs in which the sciatic nerve had been divided; while fully developed attacks were usually obtained between the 20th and 27th days (earliest 5th, latest 71st day). Horsley, in his *Brown Lectures* (abstract, *Lancet*, ii., 1886, p. 1212), suggests that racial differences may possibly account for the longer incubation period which has been found to elapse in this country.

pinch in this respect. We were never able to bring on an attack by blowing on or stroking the skin of the epileptogenous zone.

In 10 of these animals at a later date (minimum 31st day, maximum 82nd day, average 56th day) a similar stimulus provoked a fully developed convulsive attack. Spontaneous fits were only observed by us in one instance, although a number of the animals were kept under observation for from four to six months after the operation.

In the remaining 30 cases the result was negative, neither scratching movements nor fits occurring.

Since a number of the animals operated on died or were killed before the time when the fits could be expected to occur, the last paragraph is necessarily somewhat misleading. In order to obtain a more correct idea as to the frequency with which scratching or fits occur, it is obvious that only those animals should be included which survived the operation for a time sufficient to allow of their occurrence. Allowing five weeks (35 days) to elapse after the operation,<sup>1</sup> we find that 41 animals survived this date, including all of those which exhibited scratching or fits. In other words, then, we may say that of these 41 animals, 28, or 68 per cent., showed scratching movements; in 10, or 25 per cent., fully developed fits were elicited; while in 13, or 32 per cent., no result was obtained.<sup>2</sup>

Our observations support Brown-Séquard's statement that there is no direct time relationship between the appearance of "scratching" and the later development of fits.

An analysis of possible factors which might account for the occurrence of scratching and fits in only a proportion of the animals operated on brought out an interesting point, to which we shall now refer.

*Relation of the Age of the Animals at the time of operation (as approximately estimated by the weight) to the subsequent development*

<sup>1</sup> We have fixed upon this time as convenient, since the 36th day was the average date on which scratching movements were first observed.

<sup>2</sup> Among 10 animals, the progeny of guinea-pigs in which this operation had been carried out, and in which "scratching" or fully developed fits had been obtained, in none were we able to produce scratching movements on stimulating the area of the epileptogenous zone, nor was there in a single instance a congenital absence of the toes, as described by Brown-Séquard. To these observations we hope to refer in a later note.

*of the Convulsive Phenomena.*—All the animals employed in this research were weighed at the time of operation. On analysing the figures so obtained it would appear that a much larger proportion of the heavier animals developed fits than was the case with those of lighter weight. Thus among the 41 animals which survived operation by 35 days, we found that 14 weighed 650 grams or more at the time of operation,<sup>1</sup> and that 8 of these took fits. Further, of the 27 guinea-pigs which were under 650 grams at the time of operation, in only two instances were fits subsequently observed. In other words, 57 per cent. of the animals over 650 grams at the time of operation took fits, while only 7·4 per cent. of the animals below that weight did so. A closer scrutiny of these results strengthens this relation, for of the 6 animals which weighed over 650 grams at the time of operation and did not take fits, all died before the 56th day (the average date of the appearance of fits). It is therefore quite possible that had these animals lived, some or all might have taken fits, for in three of the ten instances in which fits occurred the dates of their first appearance were the 68th, 74th, and 82nd day respectively. In other words, we found that in all the animals which weighed over 650 grams at the time of operation and survived the 56th day, fits were subsequently elicited.

While of the 27 animals which were under 650 grams in weight at the time of operation, although as many as 21 survived the 56th day, yet in only 2 (9·5 per cent.) were fits obtained. With regard to these two animals—in one case, that of an animal which weighed 400 grams at the operation, a somewhat doubtful fit, of unusually short duration, was obtained on one occasion only, there being no liability to attacks persisting over some weeks, as was the general rule. As to the second case, it is perhaps significant that in this animal, which weighed 493 grams at the time of operation, fits were first elicited when it reached a weight of 700 grams. In this connection we may mention that 6 animals which when operated on weighed under 650 grams subsequently exceeded that weight, but that with this one exception, in none were fits produced.

<sup>1</sup> 650 grams may be taken as the approximate minimum weight of the adult guinea-pig. Article by Ch. Livon, "Le cobaye," *Diction. de Physiol.* (Richet), vol. iii., 1898, p. 902.

These results are summarised in the following table :—

*Table to illustrate the relation between the Weight of the Animals at the time of operation and the subsequent development of Brown-Séguard Fits.*

Weight	ANIMALS SURVIVING OPERATION BY 35 DAYS			ANIMALS SURVIVING OPERATION BY 56 DAYS		
	Number	Fits	Percent.	Number	Fits	Percent.
Above 650 grams	14	8	57	8	8	100
Below       ,,	27	2	7·4	21 <sup>1</sup>	2	9·5
	(or perhaps 1		3·7)	(or perhaps 1		4·7)

These figures, though they are not based on a sufficiently large material to warrant an absolute conclusion, are interesting, since they appear to indicate that the adult guinea-pig is more susceptible to the occurrence of Brown-Séguard's fits as a sequel to excision of a portion of the sciatic nerve than is the animal which has not attained full growth.

Sex, operation sepsis, and the appearance of trophic changes in the paralysed limb were all considered as possible factors predisposing to the occurrence of fits, but as the results were entirely negative they may be dismissed without further comment.

Since the animals operated on were among batches obtained from dealers in various parts of the country at different times, the possibility occurred to us that family predisposition might afford an explanation of these results. Enquiry into this possible source of fallacy, however, justifies us in excluding it in the present series.

Brown-Séguard states that animals which are very well fed and confined in a small space are specially prone to convulsive attacks. We have made no observations bearing on this point.

An analysis of our material showed that the variations in weight in the animals in which fits occurred were not constant. In some a fall in weight was observed before the fits made their appearance, while in others a rise was noted.

We were inclined to think that temperature seemed to

<sup>1</sup> In 16 of these animals it was possible to produce scratching.

influence the occurrence of the attacks. It was observed that in the majority of cases in which fits occurred, a few weeks after their appearance the tendency to them would disappear, yet in some cases if the weather were very cold it might again reappear. We do not wish, however, to speak with dogmatism on this point, which may have been merely a coincidence, until we are in a position to bring forward definite data.

*Summary.*—A small portion of one sciatic nerve was excised in 41 guinea-pigs, all of which survived the operation by at least five weeks.

In two-thirds of this number (28), "scratching movements" were obtained on forcibly pinching or applying a fairly strong Faradic current to the epileptogenous zone on the face and neck of the same side; in a quarter (10), fully developed fits were elicited at a later date; in a third (13), neither scratching movements nor fits were obtained. In only one case were spontaneous convulsions seen.

The average date for the appearance of the scratching movements was the 36th day, for the fully developed fits the 56th day.

The age of the animal at the time of operation (as approximately judged by the weight) appears to be a factor of importance in determining the subsequent development of the fully developed Brown-Séquard fit. Thus in all of the animals (8) which weighed over 650 grams at the time of operation (this figure was selected since it corresponds to the average minimum weight of an adult guinea-pig) and which survived the 56th day (the average date at which the first fit was noticed), fits were obtained; whereas in only 2 of the 21 animals which weighed under 650 grams at the time of the operation and survived for the same period were we able to induce them.

Is it possible that the variable results obtained, for instance, in Obersteiner's laboratory, are to be explained by supposing that because of the present increased demand for guinea-pigs in Vienna a smaller percentage of full-grown animals is now obtained for experimental purposes than was the case thirty years ago?

Sex, operation sepsis, the time of appearance of trophic changes in the paralysed leg, and family predisposition were carefully considered as possible factors in relation to the occurrence of fits in the present series with a negative result.

## Abstracts

### ANATOMY.

**ON THE INTRINSIC FIBRES OF THE CEREBELLUM, ITS (381) NUCLEI AND ITS EFFERENT TRACTS.** R. H. CLARKE and Sir VICTOR HORSLEY. *Brain*, Spring 1905, p. 13.

LOCALISED lesions of the cerebellar cortex in the vermis and lateral lobes were made in twenty-three animals—cats, dogs, and monkeys—in some cases the nuclei being injured as well. The resulting degenerations were traced by the Marchi method (Busch's modification). The authors have arrived at the following general conclusions:—

1. No fibres issuing from the cerebellar cortex enter any of the peduncles; all fibres leaving the cerebellum by way of the peduncles have origin in one or other of the cerebellar nuclei.

2. From any given area of the cortex, fibres could be traced to one or more of the cerebellar nuclei of the same side; no cortico-nuclear fibres were found to cross the middle line.

The *roof nucleus* is connected practically with all parts of the cortex, and must therefore be functionally the most important. Fibres were traced from it to the vestibular nuclei (Deiters and Bechterew), to the grey matter surrounding the superior cerebellar peduncle, and to the locus cœruleus.

The *nucleus globosus* is only connected with the cortex of the vermis and not with the lateral lobe or flocculus.

The *nucleus dentatus* is intimately associated with the pennate lobules, but not with the flocculus and paraflocculus. The only connection between this nucleus and the vermis is with the three anterior lobules of its middle lobe.

The intrinsic arrangement of the cerebellar cortex and nuclei may be regarded as consisting of two distinct organs, a spino-cerebellar, and a cerebro-cerebellar looped system, of which the parts are thus arranged:—

(a) *Spino-cerebellar*.—Spinal cord (Clarke's col.)—tracts of Gowers and Flechsig—cortex of vermis—roof nucleus—vestibular nuclei—vestibulo-spinal tract—anterior cornua of spinal cord.

(b) *Cerebro-cerebellar*.—Cerebrum (temporal lobe chiefly)—crus cerebri—pontine nuclei—middle peduncle—cortex of lateral lobe—dentate nucleus—superior peduncle—red nucleus and thalamus.

The authors are of opinion that it is the temporal lobe of the cerebrum which is most directly connected with the cerebellum and not the motor cortex.

3. With regard to the arcuate or association fibres linking

together the folia one with another, very few were found to pass from the vermis to the lateral lobes. They pass laterally only to the second folium from the edge of the lesion, rarely as far as the third, but antero-posteriorly to a much greater distance. Extremely few arcuate fibres pass to the nodulus. The disposition of the arcuate fibres supports the view that the vermis and lateral lobes are independent of each other.

4. The fibres of the different groups differed in size. The *cortico-nuclear* fibres were medium and fine; *arcuate*, fine; *nucleo-peduncular*, large (sup. peduncle), fine (middle), medium (inferior).

SUTHERLAND SIMPSON.

#### ON A HITHERTO UNDESCRIBED NUCLEUS OF THE RHOMBENCEPHALON.

(382) **BENCEPHALON.** [Di un nucleo non descritto del Rom-bencefalo (Nucleo superiore del corpo restiforme).] A. BIANCHI, *Riv. di Patol. nerv. e ment.*, Vol. x., Sett. 1905, p. 423.

THE author has examined by various histological methods the cells lying in or around the upper end of the corpus restiforme. Both he and Tkacenko had previously described a special nucleus in connection with this tract—named by the latter author nucleus superior corporis restiformis—and the present paper is only a confirmation of the earlier descriptions.

In man, masses of grey matter lie in connection with the corpus restiforme; they are split up into three separate masses: two lie at the level of the 8th nerve, the other at the level of the 5th. They are visible from the fourth month of intra-uterine life, and are not identical with any hitherto described nucleus, except that referred to by Tkacenko. As it has not been identified in any animal except man, its exact relations cannot be determined by experiment, but possibly its cells are intercalated neurones in the direct cerebellar roots of the cranial sensory nerves or in the direct cerebellar tract.

GORDON HOLMES.

#### PIGMENTATION OF THE PIA MATER, WITH SPECIAL REFER-

(383) **ENCE TO THE BRAIN OF MODERN EGYPTIANS.** W. St S. SYMMERS, *Journ. of Anat. and Physiol.*, Vol. xl, New Series, Vol. i., 1905, p. 63.

THIS condition consisted in a pigmentation of the pia, varying from smoky grey to jet black, and most apparent on the lumbar and cervical enlargements, on the bulb, about the peduncles, and over the base of the brain, especially on the optic chiasma and orbital convolutions. From two hundred successive post-mortem exami-

nations made in Cairo, the author found that apparently the pigmentation was a normal occurrence, not related to age, sex, or cause of death, and that it was particularly marked in the case of Egyptians, although it also occurred among Europeans. It is also a condition which is found in the lower animals.

DAVID WATERSTON.

**THE CIRCLE OF WILLIS.** FAWCETT and BLACHFORD, *Journ. of* (384) *Anat. and Physiol.*, Vol. xl., Third Series, Vol. i., 1905, p. 63.

IN 700 specimens examined, the circle was complete in 96·1 per cent. and incomplete in 3·8 per cent. It was symmetrical in 73·4 per cent., and was both complete and symmetrical in 72·8 per cent.

Incompleteness was due to absence of one or both posterior communicating arteries, and only very rarely to absence of the anterior communicating—*i.e.* in ·14 per cent. Asymmetry may consist in the absence of a posterior communicating artery, in doubling of an anterior cerebral—this is more common in males than in females—or in the origin of one or both of the posterior cerebral arteries from the internal carotid.

The anterior communicating artery was found doubled in 7·2 of the cases, and in the proportion of 32 female cases to 19 male.

DAVID WATERSTON.

**ON THE DEVELOPMENT OF THE HIND-BRAIN OF THE PIG.**  
(385) O. CHARNOCK BRADLEY, *Journ. Anat. and Phys.*, Vol. xl., 1905, p. 1.

THIS communication forms the first part of a paper in which are stated the results of an investigation—aided by reconstruction models—into the developmental history of the hind-brain of embryos ranging from nineteen to seventy days old. In the youngest material the hind-brain is remarkable for its simple and uniform architecture. Transverse sections taken anywhere, except in its most anterior part, are strikingly similar in appearance. Although alar and basal laminae can easily be detected in the spinal cord and in the mid-brain, there is no possibility of recognising them in the hind-brain of the youngest embryo used in the research. As development proceeds, however, the groove between the two laminae extends forwards from the spinal cord into the posterior part of the hind-brain. But at no time can it be said that distinct alar and basal laminae are present anterior to about the middle of the ventricle.



It has already been pointed out in this Review ("Neuromeres of the Rhombencephalon of the Pig," *Rev. Neur. and Psych.*, vol. ii.) that the hind-brain of the pig at one period of its development exhibits seven neuromeral depressions. Further investigations has demonstrated the persistence of these grooves for a longer time than is usually recognised. And, further, it seems probable that the third groove is the direct antecedent of the anterior fovea of the floor of the fourth ventricle of the adult brain.

In connection with the cerebellum, the material examined lends support to the conception of its origin from two lateral *Anlagen*, joined in the middle line by a connection originally thin. As a consequence of the growth in thickness of the two rudiments, without a corresponding thickening of the median connection, a deep antero-posterior fissure is produced, which develops into a veritable cerebellar ventricle, such as has been already described in mammalian embryos by Blake.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

**THE CENTRAL GUSTATORY PATHS IN THE BRAINS OF**  
(386) **BONY FISHES.** C. JUDSON HERRICK, *Journal of Comp. Neurol. and Psychol.*, Vol. xv., 1905, p. 375.

THE Teleostean fishes possess taste buds scattered over the mucous surfaces of the mouth, gills, and lips, but some groups have, in addition, the so-called "terminal buds" in the lips and outer skin. It has been previously shown that such fishes do in reality taste with these cutaneous taste buds. The peripheral innervation of all these organs has been already definitely determined, and the present paper represents an attempt to correlate the peripheral gustatory components with their central conduction paths and centres.

It is known that the taste buds of the mucous surfaces are innervated by the sensory roots of the 7th, 9th, and 10th cranial nerves which terminate in laterally placed enlargements of the bulb known as the lobus vagi. The cutaneous taste buds are, on the other hand, supplied exclusively by the sensory 7th, and where they are numerous, as in the Cyprinoids and Siluroids, another dorsally and medially placed unpaired enlargement of the bulb, the lobus facialis or tuberculum impar, appears, in which these nerve-fibres alone terminate. The fibres from both the mucous and cutaneous taste organs are associated with unspecialised visceral fibres, and this mixed system is known as the communis

system. It is not yet possible to make a complete analysis of this communis system, but by the comparative method the chief secondary connections of both types of fibres has been determined with considerable probability.

*Primary gustatory centres—lobus vagi.*—The root fibres of the communis system of 9th and 10th and part of 7th end in the periphery of the lobus vagi round the dendrites of the intrinsic neurones of the lobus, and round those of the cells which give off the long fibres of the secondary gustatory tracts. The intrinsic neurones diffuse the afferent impulses to the motor nuclei of the bulb, to the substantia reticularis, which is the chief medium of communication between the sensory and motor centres for simple reflex paths, and sends fibres into the descending secondary communis tract. At least one intermediary element is always interposed between the peripheral gustatory neurone and the peripheral motor neurone. In the Cyprinoids, in which there is a well-developed palatal organ, the inner portion of the lobus vagi is occupied by a motor layer—probably derived from the nucleus ambiguus and related to the dorsal vagal nucleus—which supplies its musculature. A small lobus glossopharangei lies oval to the lobus vagi. It receives communis fibres from 7th and 9th, and has the same structure as the lobus vagi.

The fibres from the cutaneous taste buds end exclusively in the periphery of the lobus 7th. This is an unpaired dorsally situated structure (also known as the tuberculum impar or lobus 5th of Mayser). It contains no motor layer, probably because the reflex act which originates in the extra-oral organs must be most extended, but some cells in its ventro-lateral surface—nucleus intermedius facialis—with which it is closely connected, send fibres to the nuclei of the motor cranial nerves.

*Secondary gustatory tracts.*—The descending secondary gustatory tract is derived from both the lobi 10th and 7th. It passes caudwards along with the spinal 5th root and ends in the inferior secondary gustatory nucleus, which is situated near the funicular (dorsal column) nuclei, and is intimately connected with the commissural nucleus of Cajal. Part of the fibres from the lobus 7th, however, enter the ventro-lateral columns of the cord, and form a path for the extensive reflex acts which result from stimulation of the cutaneous taste buds.

The ascending secondary gustatory tract also receives fibres from the vagal and facial lobes. It passes oralwards ventral and mesial to the spinal 5th root and ends, partly after decussation in a superior commissure in the superior secondary gustatory nuclei. The latter are situated in the dorsal part of the isthmus, and probably correspond to the Rindenknöten of Mayser. It must be regarded as a specialised portion of the substantia reticularis,

which is displaced dorsalwards by the advent of other lateral structures. As it is closely connected with the nucleus valvulæ cerebelli and with other nuclei in its neighbourhood, it can act as a centre for more complex and better co-ordinated gustatory reflexes.

*The tertiary gustatory tracts.*—These spring from the secondary gustatory nucleus and pass to the lobus inferior intermixed with the cerebellar tracts for the same destination. This is the highest known gustatory centre, and within it gustatory and olfactory sensations must become associated.

From it springs the tractus lobo-bulbaris, which ends chiefly in the substantiæ reticularis of the bulb, though some of its fibres may enter the cord. It is the main path for descending impulses from the lobus inferior, and is probably the chief motor return path from the higher gustatory and olfactory centres.

The discussion of the mammalian homologies of these structures had better await more exact acquaintance with the nervous anatomy of the intermediate and the higher vertebrate classes.

The paper represents work of exceptional excellence. The descriptions are clear and concise and well illustrated by figures.

GORDON HOLMES.

**ON THE TOPOGRAPHY OF THE MOTOR AND SENSORY  
(387) FIBRES IN THE MIXED NERVE.** (*Sulla topografia  
delle fibre motrici e sensitive nei nervi misti.*) FRANCESCO  
FRANCESCHI, *Riv. di Patol. nerv. e ment.*, Vol. x., Sett.  
1905, p. 401.

THE author first refers to the work of Viennay, who has stated that in the peripheral nerves the sensory fibres run in the central portion of the nerve trunk, while the motor fibres lie at the periphery. He then describes his experiments on dogs, in some of which he has removed the posterior root ganglia, and in others cut the anterior roots, in various regions of the cord.

These experiments have led him to the following conclusions:—

1. In the mixed peripheral nerves of the trunk and of the limbs of the dog, neither exclusively motor nor exclusively sensory bundles exist.

2. The numerical relation between the motor and sensory fibres is equal in all the bundles, but has nothing to do with the central or peripheral portion of the nerve trunk.

3. In every bundle the motor and sensory fibres have a uniform and promiscuous distribution throughout the nerve, in such a way as to exclude any systematic arrangement of the fibres.

4. In young dogs the degeneration is never so conspicuous as in the adult dog, because the absorption of the degenerated myeline proceeds so rapidly.  
R. G. Rows.

**ON CONDITIONS AFFECTING THE MAXIMAL RATE OF**  
(388) **VOLUNTARY EXTENSOR AND FLEXOR MOVEMENTS**  
**OF THE RIGHT ARM.** ROBERT HARVEY GAULT, *Am. Journ.*  
*of Psych.*, July 1905, p. 356.

THIS paper is the outcome of an elaborate series of experiments on fourteen subjects. The chief results may be thus briefly summarised. With but two indecisive exceptions, the fourteen subjects were found to be more rapid in flexor than in extensor movements, but the mean variation of the flexor times was greater than that of the extensor times.

The rate increases and the mean variation decreases as the running start is made longer, but not beyond a certain maximal start. The greatest loss in time and the largest variations occur within the first centimetre of the course, perhaps within the first half centimetre.

For some subjects there is, as a result of practice, a greater increase in the rate of extensor than of flexor movements.

The initial backward start or "back pressure"—*i.e.* the pressure of the hand or arm against the post from which the movement is started—is not a constant factor in determining the rate of movement. The greater amount of "back pressure" almost invariably occurs, on the average, in preparation for flexor, which are the more rapid movements.

The paper includes three figures showing the arrangement of the apparatus made use of for the experiments.

W. B. DRUMMOND.

## **PATHOLOGY.**

**A CASE OF ABSENCE OF CORPUS CALLOSUM.** By D. DOUGLAS-  
(389) CRAWFORD, *Journ. Anat. and Physiol.*, Oct. 1905, p. 58.

THE author describes a case of absence of corpus callosum in an elderly male whose previous history could not be obtained. The corpus callosum is represented by a round bundle of fibres, half an inch in diameter, immediately anterior to the anterior pillar of the fornix. Both hemispheres are richly convoluted. On the mesial surface there are to be noted: (1) the short gyrus fornicatus; (2) the complete separation of the anterior and posterior limbs of

the calcarine fissure by the cuneo-lingual gyrus appearing on the surface; (3) the radiate arrangement of the sulci.

On the external aspect there is an unusual subdivision of the superior and middle frontal convolutions, and great difference in shape between the right and left ascending frontal convolutions. The left fissure of Rolando is forked at its upper end. Certain gyri, especially the angular and posterior parietal, are irregularly indented by shallow sulci, which radiate from a point just behind the posterior limb of the Sylvian fissure.

A narrow band could be traced from a point in front of the anterior commissure, where it was continuous with the olfactory tract, along the dorsal surface of the rudimentary corpus callosum, backwards in immediate association with the fornix, to arch upon the posterior end of the thalamus and terminate in the uncus. This marginal band evidently represents a complete limbic lobe.

The fornix has not united with its fellow of the opposite side. White fibres pass from different portions of the cortex into this limbic lobe. The fibres of that portion of corpus callosum present connect only the anterior portions of the frontal lobes.

The writer has not been able to trace any cases since the reviewer's paper in 1888. The following important papers (and there may be others) have appeared since then: Zingerle (*Arch. f. Psychiat.*, Bd. 30, 1897); Marchand (*Berl. klin. Wochenschr.*, No. 8, 1899); Jelgersma (*Arch. f. Psychiat. u. Nervenheilk.*, Bd. 32, 1898); and Arndt and Sklarek (*Arch. f. Psychiat.*, Bd. 37, 1903).

ALEXANDER BRUCE.

**DISPLACEMENT OF PYRAMIDAL FIBRES INTO THE POSTERIOR COLUMNS IN MAN.** (Ueber die Verlagerung von Pyramidenfasern in die Hinterstränge beim Menschen.)  
BUMKE, *Neurolog. Centralbl.*, Oct. 16, 1905, p. 938.

THE author briefly records two cases showing that fibres of the cortico-motor tracts may, in rare instances, be displaced into the posterior columns of the spinal cord in man—a relation analogous to the position of fibres in certain of the lower animals.

In the first case the lesion was softening in the internal capsule. In the posterior columns, at the lower part of the medulla, and at the upper cervical region of the spinal cord, a few degenerated fibres were seen on the side opposite to the lesion, close to the posterior grey matter, in addition to the usual degeneration in the crossed pyramidal tract. At the level of the first cervical nerve root these fibres began to pass through the posterior horn of grey matter. At the second cervical segment they had passed into the crossed pyramidal tract.

The second case was one of old softening in both cerebral hemispheres. The pyramidal fibres of both sides were degenerated. At the decussation in the medulla an irregular bundle of degenerated fibres was seen in front of the central canal. At a lower level this bundle divided into two sections. At the level of the accessorius nerve the degenerated fibres were situated close to and in front of the posterior commissure; they were only separated by a fine zone of grey substance from the posterior columns; they then passed laterally through the posterior horns into the lateral pyramidal tracts.

R. T. WILLIAMSON.

**MICROGYRIA.** PAGE MAY, *Brit. Med. Journ.*, ii., Oct. 1905, (391) p. 1100.

IN the Pathological Section at the British Medical Meeting at Leicester, Dr Page May demonstrated a series of life-size sections of a brain of a case of microgyria. The material had been studied by making methylene blue preparations of certain portions of the spinal cord and of the cerebral and cerebellar cortex, whilst the remainder of the brain and most of the cord were cut in serial section and stained by the Weigert-Pal method. The term microgyria is (which commits one to no causal theory) preferable to that of the usually adopted one of hemi-atrophy; because in most of the cases, if not in all, the condition arises from arrested development (hemiagenesis) in early life and not from atrophy of an already mature brain. The usual cause of this is vascular occlusion.

Three adult cases somewhat similar to the present one, which had been published by Drs Mott and Tredgold in *Brain* (1900, Part xc.), were referred to, and also one occurring in infancy and published by Dr Batten in the same volume. The incidence of the disease, however, in all these cases was on the left cerebral hemisphere, and similarly also in a very pronounced case (not yet published) by Dr Charlton Bastian. Drs Mott and Tredgold divided the cases roughly into two classes: (1) Cortical—in which the original lesion is confined to the motor cortex or underlying white matter; and (2) Basal—in which the lesion is situated in the structures at the base of the brain. The present case seemed to be intermediate between 1 and 2, for the cerebral cortex and basal ganglia, judging from the secondary and tertiary changes in other parts of the central nervous system, were about equally and simultaneously affected.

The clinical symptoms presented by the case were briefly those of left hemiplegia and progressive dementia associated with epileptiform convulsions.

Inspection of the brain at once showed that the right cerebral

hemisphere was less than two-thirds the size of the left, which was normal; whilst the opposite (left) half of the cerebellum was notably smaller than the right. The stress of the disease fell chiefly on the right centro-parietal, less so on the frontal, and least on the occipital region. These areas were smaller and thinner than on the healthy (left) side, and were wanting in both grey and white matter. Dr Watson, who has for many years at Claybury made a special study of the histological characters of the cortex, kindly examined some of the methylene blue sections and stated that in the affected cortex all layers were thinner than normal, but especially the pyramidal layer and the inner line of Baillarger. The large motor (Betz) cells were greatly reduced in number and size, whilst those remaining were abnormal in appearance. In the ascending parietal the granular layer was the one least affected, whilst in the visuo-psychic area and frontal region the pyramidal layer was the one most affected. Other notable changes in the right side of the brain were a very much smaller pyramidal tract throughout, and a somewhat reduced mesial fillet, whilst the right optic thalamus and adjoining structures were smaller than normal. The right lateral ventricle was moderately dilated.

Whereas the right side of the cerebellum was unaltered, the left was distinctly smaller than normal, and its cortex showed a marked diminution in the size and number of the Purkinje cells, and some wasting of the granular layer. Moreover, the left dentate nucleus and the superior cerebellar peduncle, were markedly small and undeveloped. Owing to the smaller size of the right side of the pons and medulla, the median raphé was displaced to the right. In the spinal cord the chief changes were diminution in number, or atrophy of the anterior horn cells on the left side, with smallness of the right direct and left crossed pyramidal tracts. The left antero-lateral columns were also smaller than normal.

AUTHOR'S ABSTRACT.

**FORMS OF DEGENERATION IN THE POSTERIOR COLUMNS**  
**(392) OF THE SPINAL CORD.** WILLIAMSON, *Medical Chronicle*,  
 Oct. 1905, p. 14.

THE author divides the degenerations of the posterior columns into (1) changes which commence in the posterior root fibres directly these fibres pass through the spinal pia mater into the cord; (2) changes in the posterior columns which do not commence at the point just mentioned.

In the first group the degeneration is seen to commence where the fibres enter the cord, *i.e.* at the point where the neurilemma is lost and where probably also the myelin sheath is also absent

for a short distance, leaving the axis-cylinder naked. This is the so-called vulnerable point. The degeneration affects the internal division of the intra-medullary part of the posterior root fibres, while the fine fibres composing Lissauer's area remain intact. From the root-entry zone degeneration is traced along the fibres passing into the grey matter, *e.g.* to Clarke's column; and if the degeneration is well marked in the lower part of the cord, then Goll's column in the cervical region shows considerable change.

The above changes have been observed in early locomotor ataxy, diabetes mellitus, intra-cranial tumour, general paralysis, chronic ergotism, carcinoma, etc.

The theories advanced as to the causation of these lesions are mentioned by the author.

In the second group of cases the posterior roots, as in the first group, are normal; but here the degeneration does not commence at the so-called vulnerable point. The root-entry zone is either not affected or only slightly so.

In one variety of this group, *e.g.* severe anæmia, changes occur in the posterior columns only or combined with lateral column changes also. The degeneration is often clearly related to the course of the blood-vessels, especially in the posterior intermediate septum. The degeneration usually increases from below upwards and often occupies only the middle three-fifths of Goll's tract.

In some cases of postero-lateral sclerosis the posterior column changes apparently have no relation to the blood-vessels, but are similar in distribution to those above-mentioned, *e.g.* ataxia paraplegia, combined postero-lateral degeneration.

Somewhat similar changes are seen in the posterior columns in Erb's syphilitic chronic spinal paralysis.

Other causes of posterior column degeneration are: pellagra, syringo-myelia, senility, amyotrophic lateral sclerosis, progressive muscular atrophy, hereditary spastic paraplegia.

In disseminated sclerosis there are sclerotic patches in the posterior columns unaccompanied by ascending changes, due probably to the persistence of the axis-cylinders.

The patches are distributed irregularly, and the posterior root-fibres within the cord are unaffected.

DAVID ORR.

**THE INFECTIOUSNESS OF ZONA. (Le Zona: sa Contagiosité.)**  
(393) BOUYGUES, *Arch. Gén. de Méd.*, Sept. 26, 1905, p. 2454.

A LADY developed a typical herpes zoster over the right side of the thorax and the upper arm, and five days later her husband developed the same about the level of the fourth intercostal space. "No zona comes alone into hospital" is a generalisation which



has some truth in it. It is a matter of observation that cases of this disease sometimes occur in runs, and one attack practically always confers immunity. Whether the primary seat of the lesion is in the posterior root ganglia or not is still *sub judice*. Perhaps cutaneous nerve filaments are the first to be affected.

S. A. K. WILSON.

### CLINICAL NEUROLOGY.

**ON MYATONIA CONGENITA (OPPENHEIM).** WILHELM KUNDT  
(394) Inaug. Diss., Leipzig, 1905.

IN 1900, Oppenheim published a note on a condition "of general or localised hypotonus of the muscles occurring in childhood," which he regarded as a new and distinct disease, and to which he gave the name "Myatonia Congenita." Since then, three short communications on the subject have appeared, two by Oppenheim and one by Spiller.

Kundt's dissertation contains a careful description of another case of the disease from Oppenheim's klinik. He also gives a *résumé* of Spiller's case, and discusses shortly the symptoms, diagnosis, and prognosis of the condition. The main facts of the case which he reports are as follows:—

The patient was a girl of 18 months, of healthy parentage, and the pregnancy and labour had been normal. She had not been noticed to move her lower limbs until she was six months old, and then it was only the legs and feet that moved a little. Even when admitted her thighs seemed quite paralysed. The arms, however, were normal and the child could sit up. Sucking and swallowing were normal and speech only slightly less than usual at her age. The eyes and other organs of special sense were normal and the intellectual development good.

The child was well-nourished and of normal size for her age; and there was a good deal of fat, especially over the pelvis and on the thighs. She lay on her back with the lower limbs rotated outwards and the knees flexed, while passive movements of flexion and outward rotation at the hip-joints were unusually free; those of extension and especially inward rotation were somewhat interfered with. Flexion of the knees was extremely free, but extension was limited. Passive movements of the feet were unrestricted. Spontaneous active movements were seen only in the toes and feet, and rarely in the left leg. Painful stimuli caused a fairly free flexion of both knee-joints, especially of the left, while extension was limited in extent. The left hip-joint could be slightly flexed and very slightly abducted on stimulation; the right not at all. The knee-jerk was absent on both sides. The Achilles-

jerk was distinct on the left and ill-defined on the right side. The abdominal reflexes were present. In the legs there was practically normal electrical reactions to both currents. In the thighs, only the flexors, and perhaps to a slight extent the adductors on the left side, reacted to faradism and indirect galvanic stimuli; the extensors, and especially the quadriceps, did not react. Direct stimulation, however, with very strong currents produced distinct active contractions. In the muscles affected there was considerable diminution of the electrical excitability but no R.D. Somewhat delayed dentition and a partially open fontanelle were the only further abnormalities noted. The muscles of the face, trunk, and arm were unaffected. The bones were quite normal on X-ray examination. Four months after the first examination the child was found to have distinctly improved.

JOHN THOMSON.

**A CASE OF MALFORMATION OF THE EAR WITH FACIAL (395) PARESIS.** (Ueber einen Fall von hochgradiger Missbildung eines Ohres mit scheinbarer Facialis-parese.) NOVAK, *Prag. med. Woch.* Sept. 28, 1905, p. 535.

THE patient was a man fifty-six years old, who in addition to obvious mental abnormalities presented a curious malformation of the right ear, the pinna being very much shrunken and distorted, and the external auditory meatus replaced by a dimple. The right side of the face looked exactly as though there were an ordinary peripheral facial palsy, but when examined it was found that it moved well enough in both upper and lower divisions.

The view adopted is that the condition in the face is one of hypoplasia, probably due to some developmental anomaly, inasmuch as the right half of the tongue, the same half of the lower jaw, and part of the right motor fifth, were similarly involved. The relation of the condition to bulbar palsy, *Kernschwund*, facial hemiatrophy, etc., is discussed. Defective development of the temporal bone will scarcely account for all the symptoms.

S. A. K. WILSON.

**APPENDICULAR NEURITIS.** (A Propos des Névrites Appendiculaires.) (396) RAYMOND and GUILLAIN, *Arch. Gén. de Méd.*, Sept. 26, 1905, p. 2453.

THIS is merely a brief note in reference to a previous paper by the authors on this subject. They disclaim the hypothesis attributed to them by Marcon, that all appendicular neurites are of necessity ascending.

S. A. K. WILSON.

**THE SYNDROME OF ASCENDING NEURITIS.** (*Le Syndrome de (397) la Névrite ascendante—Clinique et Expérimentation.*) SICARD, Congrès des Médecins Aliénistes et Neurologistes de France. Rennes, August 1905. (Rennes: Francis Simon.)

THIS communication, presented by Sicard to the Congress of Rennes, is remarkable alike for the completeness of its clinical and experimental research, and for the scientific spirit that animates its criticism and its conclusions. The tendency of the author is to exclude mercilessly many cases hitherto accepted as falling into the category of ascending neuritis, and to emphasise the real rarity of its occurrence and its significance from the point of view of the resistance of the organism.

A glance at the literature of ascending neuritis of the years 1820 to 1860 will serve to illustrate the altogether inordinate importance once attached to this doctrine in neuropathology. It was invoked to explain everything; sympathetic or reflex paralysis from renal, uterine, or intestinal inflammation, urinary paraplegia, meningitis or myelitis consecutive to pleurisy, etc., were terms commonly employed by the masters of medicine of those days. The reaction came with Leyden, Axenfeld, Laveran, and others. Möbius declared neuritis of this kind to be a myth, a pure fancy. At present the whole question is somewhat obscure, and most recent writers on the subject express themselves with considerable reserve.

Uncertainty is so frequently the result of inaccurate terminology that no apology is needed for the author's definition that ascending neuritis is a regional syndrome, slow, progressive, ascending from the periphery to the centre, though capable of regression and of cure; a syndrome attributable to involvement of the nerve or nerves supplying the injured segment, and characterised most usually by paroxysmal pains of peculiar severity. By this definition are excluded, if it be rigorously adhered to, degenerative alterations in nerve trunks after lesions, after amputation of a limb, etc., Landry's paralysis, anterior motor neuronitis (Grasset), interstitial hypertrophic neuritis (Djérine), as well as ascending toxic infections, as in rabies and tetanus. These will be referred to again.

The origin of the condition is constantly associated with a local infection of some ordinary bacillus, introduced probably by an injury, and the local reaction may be slight or profound. Clinically, three stages may be differentiated. The first consists of the obligatory peripheral reaction of limited extent. The second is of more or less exceptional occurrence, and is characterised by reaction *à distance* and ganglionic diffusion. Lastly, there may be an ultimate radiculo-medullary phase.

The most striking feature in the clinical picture is the acuteness of the pain that is never awanting. It very frequently manifests itself in paroxysms of intolerable intensity, always radiating peripherywards. Pressure on the nerve trunks above the affected area is seldom dolorous. Vaso-motor phenomena are met with in most cases. Objective alterations in sensibility are naturally difficult to determine. Motor disturbances are not proportionate to the sensory changes, and are usually confined to muscular atrophy in the territory of the nerve trunks affected. The spread of the disease may reveal itself by a reaction *à distance*, with radiation of the pain and additional motor symptoms such as spasms and tremors.

If this second stage is rare, the third, of radiculo-medullary involvement, is, to say the least, problematical. Cases of so-called traumatic syringomyelia are never ushered in by the obligatory peripheral stage of neuritis and pain, and this is a cardinal point in the diagnosis. It is much more probable that the inflammation, or the injury, or whatever it is, stimulates a latent gliosis to action, and is therefore an accidental and not a determining factor.

According to the situation of the exciting cause, regional ascending neuritis may be divided into two groups: (1) distant, that is to say, where the original lesion is at some point distant from the centres; and (2) proximo-central, where it is near them. Of the former, a typical instance is ascending neuritis consecutive to wounds of the hands or fingers; an example of the other is trigeminal neuritis following on dental or gingival infection. No single accurate anatomico-clinical observation exists that would justify belief in the possibility of the neuritic process spreading uninterruptedly and invading successively ganglion, root, and cord.

A partial explanation of this statement is afforded by a consideration of the anatomy of the radiculo-medullary junction. It has been definitely established that the peri-fascicular and inter-fascicular spaces of the peripheral nerve trunks form lymphatic canaliculi whose intraneural course is the exceedingly limited one of a few millimetres, and which then pass out at right angles to join the lymph spaces in the cellular tissue round the nerve. This disposition explains the quasi-impossibility of bacilli spreading and scattering in its interior. But further, the cord itself is even more protected against invasion from the side of the roots. Sicard and Cestan have shown experimentally that there is no free communication between the peripheral nerve on the one hand, and the posterior root ganglion and the cord on the other. Coloured solutions injected at high pressure into the cerebro-spinal fluid never reach the peripheral nerves; and reciprocally, similar injections passed into the trunks just distal to the ganglia fail to

penetrate the conjoined nerve (where the anterior and posterior roots run together between the dura mater and the ganglion), much less the cord itself. In fact, there seems to be at this spot a real barrier preventing the migration of cellular bodies, of bacilli, of mechanical particles, from the subarachnoid space to the periphery, or *vice versa*.

From the histological point of view, we find the usual perineuritic and endoneuritic diapedesis of leucocytes, with more or less obvious destruction of myelin sheaths and axis-cylinders, over a section of the nerve trunk of about two or three centimetres' length. If the process pass into a chronic stage, we shall notice the ordinary proliferation of connective tissue, with disappearance of nerve tubes and hypertrophy of the trunk. As far as the minute anatomy of possible changes of a radiculo-medullary nature are concerned, our ignorance at this moment is profound.

It is practically certain that the disease cannot be described as one continued ascending microbic infection, and several hypotheses have been imagined to account for the symptoms. The likelihood is that the cellular tissue round the nerve reacts to the inflammation in an ascending direction, although it is possible that the elements within the nerve itself play a similar part. Any subsequent ganglionic change is most probably due to a reaction *à distance*, and not to the course of an uninterrupted sclerotic process along the nerve and its advent at the ganglion. Excessive peripheral stimulation results in unwonted activity of centres, and a sort of posterior sensory neuronitis may be produced.

It is impracticable in this résumé to enter into details of treatment; we must be content with noting that Sicard finds deep hypodermic injections of cocaine or stovaine of remarkable value.

The second part of the communication is taken up entirely with the results of numerous experiments on animals in the attempt to infect the spinal cord from the periphery. This section of the work was carried out in collaboration with Bauer. Speaking generally, the experimental confirms the clinical. In the dog nothing is easier than to produce an ascending neuritis of limited extent, but all efforts to develop distant lesions were quite unsuccessful. It is true that in certain cases of subacute neuritic infection, determined by the intranervous inoculation of virulent bacilli, diapedetic and hæmorrhagic reaction advanced along the nerve towards the cord, but the whole limb was involved, not merely the nerve trunk, and death rapidly ensued. In every other experiment, even where the animal survived as long as six or seven months, neuritic processes created by the injection of aseptic particles, of bacilli, or of toxins in the interior of the nerve, have been very restricted in their evolution, and have never attained the medullary parenchyma.

So far then from being a plea in favour of the ready infection of nerves and propagation of the neuritic process in an ascending direction, the communication is calculated to keep these facts within their proper limits, and to throw light on the mode of protection against exogenous infection, and the defensive power of the nerve centres in view of attacks from the periphery.

S. A. K. WILSON.

**ACUTE ANTERIOR POLIO-MYELITIS IN A YOUTH.** THEODORE  
(398) A. HOCH, *Journ. of Nerv. and Ment. Dis.*, Vol. xxxii., No. 9,  
p. 545.

IN this paper a case of acute anterior polio-myelitis is described in a boy aged 16 years. The onset of paralysis occurred during the month of August, on the fifth day after the feeling of malaise. The paralysis first affected the legs, and on the following day the arms were affected. By the middle of September some improvement had taken place, but two months later the boy rather suddenly became cyanosed, with extreme dyspnoea and signs of consolidation of the right lung.

The boy died 36 hours later.

At the autopsy the cerebro-spinal fluid was clear. The cord appeared somewhat smaller than normal, and on section the grey matter was of a light pink colour, and showed considerable injection and softening in the region of the anterior horn area.

Microscopically the cord showed on section areas of softening in the region of the grey matter of the anterior horns, extending from the sacral to the cervical regions. The pathological condition was bilateral and fairly symmetrical, but groups of cells involved in one segment remain partially intact in another. The blood-vessels of the ventral grey matter showed extensive changes. They were engorged with red blood cells and the walls are thickened. Many new vessels were formed, and in places the nervous elements were entirely replaced by a fibrillar network and distended capillaries. Branches of the anterior spinal artery and of the antero-lateral pial vessels were about equally affected. These vessels appeared to lead directly into the area of destruction within the grey matter, which may be considered partly as areas of anæmic necrosis. The perivascular spaces of the pial and anterior spinal arteries leading towards these lesions were crowded with extravasated cells of various types, but only after they entered the grey matter. The larger and thicker vessels were surrounded by a round or oval-shaped cell, larger than a plasma cell, and filled with fat or myelin detritus, as shown by Marchi staining.

The antero-spinal vessel was never involved until it penetrated

into the grey matter, but the pial vessels showed a slight perivascular lymphocytic infiltration within the white matter, increasing in intensity as they neared the grey matter.

In some sections the anterior spinal branches appeared normal, and the patches of necrosis and scar tissue seemed wholly dependent upon the disease of the peripheral vessels.

The anterior horns were almost devoid of nerve cells and fibres. Those cells which remain were seen chiefly in the extreme periphery of the grey matter.

The anterior roots showed well-marked degeneration by the Marchi method.

The final portion of the paper gives a review of the disease generally. The usual etiological factors are quoted: age, the summer months June to September, and rather more stress is laid on trauma than usual.

The occurrence of the disease in epidemic form is fully dealt with, a short account being given of no fewer than eighteen epidemics.

With regard to bacteriology, no uniform results have been obtained. There is as yet no proof that the disease depends upon the action of some specific micro-organisms, but sufficient evidence has been accumulated to show that it is dependent upon toxins of various kinds, either bacterial or other poisons.

The writer summarises his conclusions as follows:—

1. Anterior polio-myelitis is the result of a primary inflammatory disease of the blood-vessels of the cord, which may be thrombotic or embolic.

2. The destruction of the ganglion cells is secondary, and depends in part upon the deficient blood supply of the diseased area, and in part upon pressure and toxins.

3. The pathological changes occurring in polio-myelitis of children and adults are apparently identical and dependent on similar causes.

4. There is sufficient evidence at hand to consider the disease as a rule of an infectious nature, not, however, depending upon a specific micro-organism, but resulting from bacterial infections of various kinds, and at times from other poisons.

5. The inflammatory changes are present in the peripheral vessels as well as in the branches of the anterior spinal artery, though these changes are seldom visible until the vessels enter the grey matter.

6. The inadequate collateral circulation within the anterior horns is favourable for sluggish circulation and embolism.

F. E. BATTEN.

**A CASE OF AMYOTROPHIC LATERAL SCLEROSIS IN WHICH  
(399) THE SYMPTOMS WERE UNILATERAL AND ASCENDING.**

CHARLES S. POTTS, M.D., *Univ. of Penn. Med. Bull.*, July-August 1905, p. 155.

IN the January 1905 number of the same journal, Dr Spiller published a series of cases of pyramidal degeneration; this case is now added as being of a somewhat similar type, in that both motor neurones appeared to be degenerated.

The patient was a hard worker who first showed symptoms at the age of 41 and came under observation six months later. The illness began with difficulty in walking, the toes of the left foot tending to catch in slight projections. Later, the left arm became weak and bulbar symptoms came on.

On examination, the eyes were found to be normal in all respects: the face showed bi-lateral spasticity, and the tongue, which was projected with difficulty, was somewhat atrophic. Speech was slurring, hesitating, and indistinct. The movements of the right vocal cord were impaired.

There was no absolute paralysis of any movement of the limbs. There was marked wasting of the small muscles of the left hand, and to a less extent of the right. The muscles generally were spastic and atrophic, and showed fibrillary tremors. Sensation and sexual power were normal, but he was very emotional. There was no lead line on the gums.

STANLEY BARNES.

**ON CHANGES OF MUSCLE SENSIBILITY IN TABES AND  
(400) OTHER PATHOLOGICAL CONDITIONS, AND ON THE  
MYOSTHESIOMETER.** (Ueber Veränderungen der Muskel-sensibilität bei Tabes und anderen pathologischen Zuständen und über den Myosthesiometer.) W. v. BECHTEREW, *Neurolog. Centralbl.*, Nov. 1, 1905, p. 978.

V. BECHTEREW finds that diminished sensitiveness of muscles to pressure is a constant symptom of tabes; it may be present even at an early stage, before disturbances of muscle-sense or of cutaneous sensation are pronounced; it appears first, and is almost always most marked in the leg muscles, but, in the ataxic stage, it is sometimes evident also in the arm muscles. Generally, but not necessarily, insensitiveness of the nerves to pressure is present at the same time, and also changes in cutaneous sensibility; but there is usually no parallelism between the diminution of muscle sensibility and defect of muscular sense.

The diminished sensitiveness of muscle and nerve to pressure



in tabes may be of importance in differential diagnosis, inasmuch as in the tabetic form of multiple neuritis—which may be confused with tabes—both nerve and muscle are usually hypersensitive to pressure.

A sketch is given of an instrument, a myosthesiometer, by means of which the degree of pressure can be regulated and accurately measured.

A. W. MACKINTOSH.

**TWO CASES OF HICCOUGH CRISES IN TABES.** (Zwei Fälle (401) von Singultuskrisen bei Tabes.) L. STEMBO *Neurolog. Centralbl.*, Nov. 1, 1905, p. 985.

STEMBO finds in the literature of tabes no mention of hiccough as a symptom or complication. He records two cases in which attacks of hiccough occurred, lasting for days or weeks or even months, greatly reducing the patient's strength, accompanied by severe pains in the region of the stomach and lower thorax, and obstinately resisting treatment.

From the long duration of the attacks and the violent accompanying pains, he believes that these attacks were true *crises*.

As regards treatment, he lays stress on washing out the stomach and feeding *per rectum*.

A. W. MACKINTOSH.

**ON SUDDEN DEATH IN TABETICS.** (Ueber den plötzlichen Tod bei (402) Tabischen.) S. GOLDFLAM, *Neurolog. Centralbl.*, Nov. 1, 1905, p. 980.

GENERALLY tabes runs a long course, and the duration of a tabetic's life is likely to be but little shorter than that of a healthy person of the same age. In rare cases, early death has occurred quickly or suddenly from some symptom of tabes, notably laryngeal crises: Goldflam has seen a case ending fatally from profuse hæmatemesis, accompanying violent gastric crises; in another case, sudden death occurred after fracture of the leg bones, probably from pulmonary fat-embolism.

But a more common cause of early or sudden death in tabes is some complication. Thus Goldflam has recently seen cases of sudden or very quick death—from cerebral apoplexy (2 cases), rupture of aortic aneurism (1 case), heart failure (3 cases, 2 of which suffered from angina pectoris), no assignable cause (2 cases). Hemiplegia is not rare in tabes: thus Fournier found it in 18 of 224 cases; usually it is transient, and it rarely leads to a fatal issue. Aortic aneurism appears to be a very frequent complication of tabes; Lesser found it in 18 of 96 cases

of tabes, Ruge in 3 of 64 cases. Heart disease, notably that affecting the aortic valves, is well known to occur frequently in tabes, but attacks of angina pectoris appear to be comparatively rare; Goldflam saw no clinical difference between these attacks in tabes and true angina which is due to changes in the coronary arteries.

It is striking that all the cases of sudden or very quick death seen by Goldflam occurred in the early stages of the disease, that the patients were all syphilitic and usually in full manhood, and that they died of complications (brain apoplexy, aortic aneurism, angina) which have a very close connection with syphilis. Such cases warn us not to be too confident in giving a favourable prognosis *quoad vitam* in tabes.

A. W. MACKINTOSH.

**THE OCCASIONAL CLINICAL RESEMBLANCE BETWEEN  
(403) CARIES OF THE VERTEBRÆ AND LUMBO-THORACIC  
SYRINGOMYELIA, AND THE LOCATION WITHIN THE  
SPINAL CORD OF THE FIBRES FOR THE SENSATIONS  
OF PAIN AND TEMPERATURE. W. G. SPILLER, *Univ.*  
*Penn. Med. Bulletin*, Vol. xviii., July-August 1905, p. 147.**

AFTER remarking on the comparative rarity of cases of lumbo-thoracic and lumbo-sacral syringomyelia, and the difficulty associated with making the diagnosis of those conditions on clinical evidence, Dr Spiller relates the case of a woman of 34 years of age in whom the symptoms suggested the possibility of a cavity in the cord in the lumbo-thoracic region. Pain in the lower limbs was followed by a slowly progressive paraplegia, the left limb being spastic and weaker than the right, which was flaccid and without deep reflexes. Both soles gave extensor responses. Dissociated anæsthesia was present on the right leg and right lower abdomen, sensibility being intact on the left side. No curvatures, no trophic changes, no muscular atrophy. The condition of the sphincters is not mentioned. The right pupil was larger than the left, but otherwise there was nothing abnormal in the trunk, upper limbs, or cranial nerves. No autopsy.

The second case reported is one of caries of the lower thoracic vertebræ in a man of 23. A slight spastic paraplegia, with a not very clearly defined dissociation of sensation in the lower limbs and the presence of genu valgum, suggested the possibility of a lumbo-thoracic syringomyelia. The discovery *post mortem* of a small tubercle in the lower dorsal part of each lateral column, together with vertebral caries and tuberculous meningitis, lends some support to the theory that the fibres transmitting thermal and painful sensations may run in the antero-lateral ascending tracts, but the notes on the sensibility of

the skin of the lower extremities indicate too much variability, and the pathological conditions are too diffuse and widespread to allow certain conclusions to be drawn.

E. FARQUHAR BUZZARD.

**ACCIDENT AND MULTIPLE SCLEROSIS.** (*Unfall und multiple (404) Sklerose.*) GROSSMANN, *Deut. med. Wochenschr.*, Oct. 12, 1905, p. 1633.

THE writer discusses at length the importance of injury as a cause of multiple sclerosis. He gives an account of the views expressed by many distinguished neurologists who have published series of cases bearing on the subject. On the one hand, several deny that the organic changes of multiple sclerosis can be produced at all by bodily injury. Thus Strümpell and Eduard Müller could trace no history of any injury in 75 per cent. of their cases, and in the remainder there appeared no connection between the injury and the disease. Also Sachs and Freund, out of 5000 cases of illness after accident, could find no instance of multiple sclerosis. Klausner, Morawitz, Pierre Marie, and Krafft-Ebing also take up this position.

On the other hand, V. Leyden believes that concussions of the body may produce the disease; Kiewlicz collected twelve cases with supposed traumatic causes; Blumreich and Jakoby assigned injury as the cause in five cases out of twenty-nine. Similarly Hoffmann, Lotsch, Schlagenhauer, Redlich, and V. Jaksch believe that they have met with cases of traumatic causation.

Others take up an intermediate position. Thus Mendel believes in a predisposition to the disease, which injury excites; Jutzler and Ziegler consider there is, to begin with, a congenital faulty arrangement of parenchyma and neuroglia. Similarly Windsheid, Gumprecht, Gaupp, and Berger have published series of cases from which they are led to believe in injury as a merely exciting cause.

The writer gives full details of a case which is most important as showing how readily an accident which merely develops the symptoms may be set down as the cause of the disease. A strong labourer, aged 28, who had previously complained of nothing, fell a short distance upon the back of his head and became unconscious. *Two days later* he was found to show the symptoms of well-developed multiple sclerosis, including double optic atrophy, nystagmus, intention tremors, and increased reflexes, which remained permanent. It is evident that the condition could not have developed in two days, and the writer urges caution in accepting a traumatic causation in other cases.

It is pointed out that in Germany and Austria a workman

would be entitled to compensation whether the injury is the direct or only an exciting cause of the disease in a man previously capable of work. A copious bibliography on the relation of injury to sclerosis is given.

JOHN D. COMRIE.

**DIVER'S PALSY.** (*Les paralysies des scaphandriers.*) BOINET et (405) AUDIBERT, *Arch. Génér. de Méd.*, Oct. 24, 1905, p. 2689.

THE authors base their observations on cases under their care at Marseilles. Diver's palsy was attributed by Leroy and Méricourt, in 1869, to hæmorrhages into the spinal cord. In a hundred divers working at depths of 30 to 40 metres, *i.e.* three to four atmospheres *above* normal atmospheric pressure, Denerouze observed five deaths and many cases of paraplegia in a period of six months. Rapid decompression is the chief cause of accident; no death occurred while the diver was under pressure. In 1882, Parissis and Tetzis described diver's paraplegia with hæmorrhage into the dorsal and lumbar regions of the spinal cord. In grave cases, pains in the shoulders and arms, with occasionally paralysis of the arms, were met with. Catsaras published, in 1888, a study of 60 cases of diver's palsy, as well as a series of experiments on animals subjected to high pressures.

Accidents are frequent amongst the sponge divers on the African coasts. The number of deaths occurring annually on board the 800 boats engaged in the Mediterranean sponge fisheries is estimated at a 100. As the divers take only one meal in twenty-four hours, fatigue plays a part in the production of illness. The paraplegia is permanent in about one-tenth of the cases, and is then usually spastic in type. Paraplegia is frequently accompanied at the onset by paralysis of the arms, which rapidly passes off. Often the spastic paraplegia ultimately predominates in one leg, diminishing in the other to such an extent that the diver can begin work again. Hemiplegia is rare, and always of short duration. The author's own cases of diver's palsy are gathered mostly from amongst the coral fishers belonging to the coast of Provence. They give account of eight deaths and thirteen cases of paralysis, ten of which are cases of paraplegia. The relative frequency of accidents is attributed to the carelessness of the divers in staying too long at great depths, not taking sufficiently long rests between dives, and submitting themselves to too rapid decompression by ascending too quickly. The authors themselves have made several descents in the diving suit. The suit used is of the ordinary type. The helmet has an inlet pipe by which fresh air from the pump enters; the vitiated air escapes by an outlet valve regulated by a lever worked by a lateral movement of the head of the diver.

The "lesser depths," so called, are from 25 to 30 metres, while the "greater depths" reach 45 metres on the coast of Provence, and 65 metres in the Ionian Sea and on the African coast. The dives are of longer duration in the lesser than in the greater depths. Coral fishing is carried on at a depth of about 30 metres. In twenty-four hours four dives at most are made, each of not more than twenty minutes' duration, with at least an hour's rest between. The diver ascends by allowing the air to inflate his suit; he can arrest his ascent by causing air to escape by the outlet valve. Rapid ascent, *en ballon*, is the chief cause of illness; the greater the depth, the more severe the resulting paralysis. The paralysis of the "lesser depths" is usually transitory; that of the "greater depths" is usually permanent, the muscles being at first flaccid, and later on in the course of the illness becoming spastic. The cases of spastic paraplegia are associated with disturbance of the sphincters, rendering the patient a chronic invalid.

The cases seen by the authors are described in detail. In the eight fatal cases, death occurred in some at the end of a few minutes, and in others not until several hours had elapsed. Unconsciousness came on almost immediately after the diver left the water, and was accompanied by cyanosis.

In the cases of paralysis, there was invariably a period of five to fifteen minutes after the diver left the water before any symptoms appeared. In some cases paralysis was preceded by a loss of consciousness of variable duration; in other cases vertigo, severe pains in the limbs and back, and tingling were observed before paralysis developed. On other occasions, hyperæsthesia or anæsthesia of the skin, increase of patellar reflex, tachycardia, and dyspnœa were noted.

As examples of rare forms of paralysis are mentioned cases of hemiplegia with facial paralysis; in one of these aphasia and delirium were also present. These patients soon recovered completely.

Paraplegia, temporary or permanent, is the commonest form of paralysis; several cases are described at length. When permanent paraplegia results, there is often at the onset of illness paralysis of the arms also, this latter rapidly disappearing. Retention of urine and fæces, with bedsores, are of frequent occurrence. Several patients suffered more than once from paralysis, having recovered from one attack and resumed work. The permanent paralysis are due to hæmorrhages into the spinal cord; this is demonstrated by autopsies. In the temporary paralyzes it is believed that no rupture of the blood-vessels by air bubbles has occurred, and consequently no hæmorrhage. The absence of autopsy leaves the pathology of these latter cases somewhat doubtful.

The pathology of the disease is that described by Paul Bert. Nitrogen is dissolved by the blood in increased quantity at high pressure, according to Dalton's law, and is liberated in the form of bubbles on decompression. These bubbles lead to rupture of the vessels and hæmorrhage results. The blocking of vessels by gas bubbles is also believed to cause infarction. Experiments on animals support these theories, and in autopsies after experiments bubbles are actually seen. The liberation of bubbles in the central nervous system leads to the most serious results, but the same phenomenon occurs in other fluids and tissues of the body.

As to means of prophylaxis, the authors give a table showing what is considered to be a safe length of time for the dive, and for the ascent at different depths; also the proper period of rest to be taken between dives. They urge that the ascent should be slow, and that the diver should rest at different levels as he comes up. The greater the depth, the shorter should be the duration of the dive, the longer the time taken for ascent, and the longer the period of rest between dives. For work at the greater depths, only one meal should be taken in twenty-four hours. In case of accident it is advised that the diver should be subjected to pressure again.

ALFRED PARKIN.

**FURTHER CONTRIBUTIONS TO LUMBAR PUNCTURE.** (*Weitere*  
(406) *Erfahrungen auf dem Gebiete der Lumbalpunktion.*) OTTO  
REHM, *Centralbl. f. Nervenheilk. und Psychiat.*, Oct. 15, 1905,  
p. 798.

THIS paper deals with the utility of the examination of the cerebrospinal fluid in cases of mental disease, and bears out the conclusions of previous observations on the constancy with which lymphocytosis occurs in syphilitic diseases of the central nervous system, and hence the value of this symptom in doubtful cases of general paralysis, and in the distinction of chronic alcoholism from various forms of syphilis of the nervous system. Rehm gives the following tabular results from examinations in which the clinical diagnosis was certain. The great difficulty is in the lack of precise information as to how far lymphocytosis may occur in syphilitic processes of other tissues than those of the nervous system. The presence of lymphocytes in general paralysis and their absence in chronic alcoholism seems to be a constant fact. Fuchs and Rosenthal in 208 cases found a positive result in 198 cases; Nissl in each of 58 cases.

Diagnosis.	Number of Cases.	Positive Result.	Negative Result.	Syphilitic history known.
Chronic alcoholism . . .	9	1*	8	...
Dementia præcox . . .	3	...	3	...
Syph. brain disease . . .	2	2	...	1
General paralysis . . .	39	39	...	12
Syph. arterial disease . . .	2	2	...	1
Atheroma . . .	3	1*	2	...
Hysteria . . .	3	2	1*	2
Idiocy . . .	2	2	...	1
Tumour cerebri . . .	2	...	2	...
Depressive mania . . .	2	...	2	...
Epilepsy . . .	3	...	3	...

\* Syphilitic infection probable or certain.

W. B. WARRINGTON.

#### THE ABSENCE OF GLUCOSE IN THE CEREBRO-SPINAL FLUID.

(407) (*De l'absence de glucose dans le liquide céphalo-rachidien.*)

DUBOS, *Annales Méd.-Psycholog.*, Nov.-Dec. 1905, p. 393.

THE writer seeks to form a conclusion on the nature of the reducing substance found in the cerebro-spinal fluid, and which is now generally considered to be pyrocatechin. He has examined the fluid by—(a) the chemical tests, including phenyl-hydrazine; (b) fermentation tests; (c) by the polariscope. He concludes that the substance is neither sugar nor pyrocatechin, but that it belongs to the purin bases, creatin, creatinin, xanthin and hypoxanthin.

W. B. WARRINGTON.

#### A CASE OF TUMOUR OF THE LEFT FIRST AND SECOND

(408) **FRONTAL CONVOLUTIONS WITH MOTOR AGRAPHIA, ETC.** J. W. M'CONNELL, *Univ. of Penn. Med. Bull.*, July-August 1905, p. 156.

GRAPHIC aphasia unassociated with paralysis of the upper extremity is so rare that the case recorded here is of great importance.

The patient was a right-handed man who at the age of 23 had a sudden convulsion after a hearty meal. Thereafter he had several major and minor attacks of an epileptic nature, none of them with localising symptoms. At the age of 28 he began to suffer from Jacksonian attacks, without loss of consciousness and

affecting the right side of the face. These attacks recurred several times a day, and were seen to commence by contraction of the muscles at the right angle of the mouth, spreading thence to the eyelids, but not to the eye muscles. Such attacks were followed by mental confusion and dysarthria for a few minutes, but there was no confusion of words in speaking.

On examination (May 1904, age 28) there was well-marked supra-nuclear paralysis of the right side of the face, much intensified after a spasm. There were no limb defects, and sensation, though a little impaired over the right side of the face, was not otherwise affected. Speech was dysarthric only. Spontaneous writing and writing from dictation resulted in a series of almost meaningless strokes, but from copy the writing was almost perfect. Some mental change had occurred (irritability, loss of memory, etc.). There was no optic neuritis.

An operation was performed, and a tumour mass was found lying across the foot of the second frontal convolution, encroaching slightly on the corresponding part of the first and third frontal and the anterior edge of the ascending frontal convolutions.

The patient made a good recovery from the operation, and within a few weeks his writing was almost perfect.

The author discusses the history of the study of graphic aphasia, and adds this case to the list of those which go to prove that the function of writing is directed by a centre which lies in the hinder end of the second frontal convolution, and which is anatomically distinct from the hand and arm centres.

STANLEY BARNES.

**PARALYSIE GÉNÉRALE ET SYPHILIS.** Communications faites (409) à l'Académie de Médecine par MM. ALFRED FOURNIER et F. RAYMOND. Masson et Cie, Paris, 1905, pp. 100.

FOURNIER discusses expressly general paralysis of syphilitic origin without entering upon the question of similar syndromes with different aetiology. He first takes up the date of onset of general paralysis with reference to the primary infection. In 65·1 per cent. of his cases the first symptoms were noticed from the sixth to the twelfth year after the chancre, there being a maximum in the tenth year. No case began earlier than two years after infection.

With cerebral syphilis, on the contrary, two-thirds of the cases began within the first five years after the infection, the largest number occurring in the third year. In all his cases of general paralysis there had been of course syphilitic infection; but out of



79 cases, with regard to which accurate information could be obtained, only 4 had been adequately treated, i.e. had been treated thoroughly for the three or four years following infection. It is this want of adequate treatment which is the important point in the ætiology; but the four patients who had been thoroughly treated show that we have as yet no sure prophylaxis against general paralysis.

In 15 cases out of 112 there were present predisposing or accessory causes, of which the most important was the strain of an exciting or ill-regulated life; in only 2 cases was there present well-marked nervous heredity, but the author does not state in what his rigorous conception of the latter consists. According to his statistics, therefore, in only a small proportion of cases was there any apparent reason why the syphilis should attack the brain. With regard to the nature of the syphilitic infection, this was invariably mild, and in only 3 out of 83 cases did tertiary symptoms appear. It is interesting that out of 243 cases of virulent syphilis observed by the author, not one developed general paralysis. According to Fournier, tertiary symptoms as a rule mean incomplete treatment. A mild attack of syphilis is more dangerous than one which by a series of manifestations jogs the memory of the patient and induces frequent periods of anti-syphilitic treatment. In order to make the prophylaxis as efficient as possible, the author recommends treatment for two years after the chancre, cessation during the next few years, which as a rule are free from manifestations, renewal of the treatment during the fifth year, and again during the seventh or eighth year after the chancre, for thus one, as it were, revaccinates the patient before the periods which statistics prove to be the most dangerous ones. It is not sufficient to prescribe mercury; over-exertion must be avoided, the life regulated, and hydrotherapy over a period of several years is most beneficial.

Raymond in his communications confirms the observations of Fournier, although he lays considerable stress on heredity as a predisposing cause. He gives in detail his reasons for looking upon syphilis as the important ætiological factor. He lays great stress upon the frequent presence of undoubtedly syphilitic lesions in general paralysis, refers to statistics, calls attention to the analogy of tabes and general paralysis and to the history of the discussion as to the rôle played by syphilis in the production of the former, and supports his views by reference to the cases of juvenile general paralysis where other factors, such as alcoholism, intellectual strain, etc., can play no part.

C. MACFIE CAMPBELL.

**PARALYSIS OF THE EXTRINSIC EYE MUSCLES IN DIABETES.****(410) (Paralysie des nerfs moteurs de l'œil chez les diabétiques.)**DIEULAFOY, *La Presse Médicale*, Nov. 4, 1905, p. 713.

IN this highly interesting clinical lecture the case taken to illustrate the subject was a man seventy-three years of age with isolated paralysis of the external rectus on the left side. The actual onset had been sudden, though preceded by constant temporo-orbital neuralgia on the same side for at least eight days. The night before the paralysis occurred the pain was particularly severe, and in the morning it was observed that the left eye was strongly converged. He complained of vertigo and diplopia, both binocular and monocular, *i.e.* he saw two images whether he regarded with both eyes or only with the left. Occasionally he even had triplopia, as when looking at the lamp hanging from the ceiling of the ward. This similarly was both binocular and monocular. No further eye symptoms were present.

The patient was neither a syphilitic nor a tabetic, but on the other hand he was a diabetic, with polyuria and polydipsia, the sugar in his urine averaging twenty-three grammes per litre. The administration of antipyrin, the application of the constant current, and the adoption of a suitable dietary, combined to effect a complete cure in three months, and at the end of three years there had been no return of the paralysis, though the diabetes persisted.

Enlisting his colleagues in a research, Professor Dieulafoy has collected fifty-eight cases of ocular palsies in diabetes, of which thirty-five concern the sixth pair. In syphilis and tabes, of course, partial or total paralysis of the oculo-motor nerves is by far the most frequent, so that the implication of the sixth has a certain diagnostic value. There is no necessary relation between the paralysis and the intensity of the glycosuria. The palsies are usually both sudden and transient, though recurrence is not uncommon. In some cases the ocular palsy may be but the prelude to a progressive ophthalmoplegia that eventually passes into a fatal polio-encephalitis.

The violent and constant neuralgia already noted is of some significance. It occurs with some frequency in the condition under consideration, and may be taken to indicate a "painful paralysis" of the nerves involved. It is conceivable that the explanation lies in the close connection of Deiters' nucleus with the oculomotor centres on the one hand, and with the descending root of the sensory fifth on the other.

The pathogeny of these diabetic paralyses is much more obscure. If it be true that diabetic neuritis is a pathological fact, whatever be the mechanism, how comes it that the motor nerves

of the limbs are spared and the motor nerves of the eye affected? And moreover diabetic sugar must be extraordinarily toxic if ocular palsies supervene in individuals with only a few grammes of sugar in their urine.

Contrariwise, it is impossible not to be struck with the unique localisation of these diabetic paralyses. In the great majority of cases the bulbar syndrome, reduced to its lowest terms, is diabetes plus paralysis of a cranial nerve, and the memorable experiments of Claude Bernard at once suggest themselves. Yet the explanation of this morbid association is to seek, for it is difficult to see how one and the same cause can produce a persistent glycosuria and a passing paralysis.

S. A. K. WILSON.

**THE MOTOR AREA OF THE HUMAN CEREBRUM, ITS POSITION AND SUBDIVISIONS, WITH SOME DISCUSSION OF THE SURGERY OF THIS AREA.** C. K. MILLS and C. H. FRAZIER, *Univ. of Pennsylvania Med. Bulletin*, Vol. xviii., July-August 1905, p. 134.

THIS paper includes a short review of the literature dealing with faradisation of the human brain, especially Lamacq's publication in 1897, and proceeds to detail about ten further contributions to the subject which have occurred in the personal experience of the authors. It is unnecessary to enter into the minutiae of these observations because they do not admit of strict scientific accuracy owing to the difficulty presented to the surgeon of determining the exact geography of a trephine whole. The result of these examinations tends to confirm the view promulgated by Sherrington and Grünbaum in regard to anthropoid apes, and by Campbell in reference to the histological character of the human cortex, that the excitable motor area is probably largely, if not entirely, pre-central in site. The authors favour the unipolar method of stimulation, and urge the necessity of a pre-arranged systematic method of procedure in making these observations for the enhancement of their scientific value.

E. FARQUHAR BUZZARD.

**CLINICAL ANATOMICAL CONTRIBUTION ON THE DISTURBANCES OF SENSIBILITY OF CEREBRAL ORIGIN.** (Anatomisch-klinische Beiträge zur Lehre der cerebralen Sensibilitätsstörungen.) KARL SCHAFFER (Budapest), *Neurol. Centralbl.*, Oct. 1, 1905, p. 888.

THE author concludes that the anatomical clinical characteristics of disturbances of sensibility of cerebral origin are as follows:—

1. This loss of sensibility is seldom complete; when complete it is due to a widespread softening destroying the whole thalamo-cortical sensory path. In such cases one gets absolute anæsthesia and analgesia in both skin and the deeper parts.

2. The loss is usually incomplete—a more or less defective sensory perception. Light touches may be scarcely or not at all felt; while heavier touches may be perceived but localised at some distance from the place touched, which seems to be characteristic of sub-cortical lesions of sensory path. This defective projection of sensory stimuli affects all kinds of sensibility—touch, temperature, and pain. The disassociation of thermal sensibility with loss of perception of warmth, while cold causes a painful sensation, is interesting to notice.

3. The variability of the intensity of anæsthesia in sub-cortical lesions is remarkable and probably dependent to a great extent on the attention paid by the patient.

4. The clinical forms of anæsthesia of cerebral origin come under three heads:—

(a) Poly-insular form—small scattered areas of anæsthesia.

(b) Hemi-anæsthesia, associated with hemiplegia, may be complete or incomplete. In the latter case it has a characteristic distribution, being more marked at the distal parts of the extremities and gradually diminishing in intensity towards the proximal parts.

(c) Total anæsthesia over whole body associated with a diplegia.

5. In some cases of hemi-anæsthesia the face is partially spared. This often seems to follow the main branches of the fifth nerve—at other times irregular areas remain free.

6. The anatomical basis of a complete and permanent cerebral hemi-anæsthesia is either an extensive softening involving all the thalamo-cortical sensory neurones or lesion of the central gyri with destruction of their terminal processes.

7. The anatomical basis of a permanent but incomplete cerebral anæsthesia (topo-anæsthesia) is a partial lesion of the tract. It is questionable if a partial lesion of the posterior part of capsule, without involvement of thalamus, can cause any cerebral anæsthesia.

8. Lesions in the anterior part of posterior capsule without any damage to the thalamus may cause a permanent and complete hemi-anæsthesia due to a lesion in the thalamo-cortical sensory path. It follows that the sensory tract arising from the ventro-lateral nucleus of the thalamus runs, mingling with the motor fibres, towards the genu. Part of it goes through the lenticular nucleus on its way to the central gyri.

COLIN K. RUSSEL.

**ON DISTURBANCES OF SENSIBILITY IN CEREBRAL DISEASE.**(413) (*Über Störungen der Sensibilität bei Erkrankungen des Gehirns.*)FRIEDRICH MÜLLER (Munich), *Sammlung klinische Vorträge*, 394, 395, p. 377.

IN several cases of lesions in the sensory paths in the brain, with, at the onset, complete hemianæsthesia and analgesia, loss of thermal sensibility and the sense of position, the author has found a gradual incomplete return of sensibility, appearing first on the face, mucous membranes of the mouth, nose, and conjunctivæ, then on the neck and trunk, always commencing at the median line. In the extremities it returns first in the proximal segments, the fingers and toes remaining anæsthetic longest. Usually some sensory disturbance remains in the arms and legs. In these cases there is often some disturbance of sensibility in the leg on the homolateral side to the lesion, pointing to the fact of its partial representation in the homolateral cortex.

Slowly progressive lesions, as tumours, cause less disturbance of sensibility, because the fibres may be simply pushed to one side—or corresponding paths on the opposite side gradually take over the functions of the damaged fibres.

If a hemianæsthesia is from the onset incomplete, it shows the same type as is seen after recovery from a complete hemianæsthesia—*i.e.* the parts of head and trunk adjacent to the mid-line are little or not at all affected, while the extremities, and particularly the hands, are most or alone affected. Here one cannot mistake the analogy to the motor hemiplegia. When a total flaccid paralysis improves, movement returns first in the face, tongue, palate, and trunk: in short, in the bilaterally acting muscles. If, however, a hemiplegia is incomplete from the onset, this same condition is present from the commencement. Anæsthesia becomes most marked in those parts where sensibility is most highly developed—whether the lesion is in the cortex, internal capsule, thalamus, or lemniscus.

The disturbance of sensibility is arranged in general at right angles to the segmental distribution. Pain sense is least impaired. Temperature and pressure sense are more so, while sense of position and the stereognostic sense are most affected.

The author has never seen a case where, with sensibility to pain, touch, pressure, and temperature intact, there has been any disturbance of localisation. The sense of localisation returns more slowly than of pain, temperature, or pressure—whether the lesion be central or peripheral—therefore he argues there are no special nerve paths and centres for localisation.

He concludes that while disturbances of sensibility due to

lesions in the cortex, corona radiata, thalamus, or tegmentum have no special characteristics by which they may be distinguished the one from the other, the more marked and more permanent the loss is, the lower down in the sensory path is the site of the lesion.

COLIN K. RUSSEL.

**THE SENSE OF VIBRATION AND ITS CLINICAL SIGNIFICANCE.**

(414) (*Untersuchungen über das Vibrationsgefühl und seine klinische Bedeutung.*) STERLING, *Deutsch. Zeitschr. für Nervenh.*, B. 29, H. 1-2, S. 57.

THIS paper adds one more to the increasing number of more or less elaborate investigations on the question of pallæsthesia. It is scarcely to be expected that repetition can be avoided, and the latest contribution is no exception. Rydel and Seiffer's diapason, the instrument adopted by the author, is once more faithfully described, and endless minutiae in reference to the physical condition of the skin, temperature, pressure, etc., are no less faithfully discussed. The hypotheses and results of previous workers in the same field are collated and criticised. He is able to corroborate the findings of Rydel and Seiffer in regard to the connection between the ataxia of tabes and the degree of diminution of pallæsthesia. Of all forms of sensation, the sensation of vibration appears to be the first affected, and in a high proportion of their cases of tabes the appreciation of the vibrating tuning-fork varied inversely with the ataxia. The two conditions, however, did not necessarily attain their maximum or minimum in the same area.

The results of the examination of all sorts of nervous cases are given. His observations seem to justify the belief that in compression paraplegia this form of sensation is affected earlier and more intensely than in any other variety of spinal cord disease. In syringomyelia there is no certain relation between the amount of touch, pain, or temperature alteration and the pallanæsthesia. The sense of vibration is constantly diminished on the paralysed side in cases of hemiplegia, even though this be the only remaining indication of the paralysis.

The conclusion arrived at after a very long discussion of theoretical and controversial matters is that all tissues in which are the end organs of peripheral nerves are capable of conducting the sensation, and it is purely a physical and not at all a physiological question which acts best. The remarkable elasticity of the relations between pallæsthesia and cutaneous and deep sensation leads the author to withhold definite opinion as to the actual clinical value of the method.

S. A. K. WILSON.

**LE SYNDROME DE MOEBIUS (AKINESIA ALGERA).** L. INGEL-  
(415) RANS, *Gaz. des Hôp.*, June 1905, p. 783.

In 1891 Moebius described under the name Akinesia Algera a new symptom-complex and gave a full report of two cases which had come under his observation. Since that time over forty similar or analogous cases have been reported. The author has collected together the literature on this subject, and by giving the details of the various cases and the comments of the various authors, he has written a short but most excellent review of the clinical relations and essential features of Akinesia Algera. Moebius described the condition as one in which it was impossible to move without pain—a state in which all movements are painful although no appreciable cause for the pain can be found. In 1895 Moebius made a further contribution to the subject in which he gave the name Dyskinesia Algera to those cases where gentle movements were still possible, and reserved the name of Apraxia Algera to cases in which speaking, writing, reading, or thinking gave rise to severe and inveterate pains in the head. He regarded the pains as being “des douleurs-hallucinations,” the pains being of an hysterical nature although the condition itself was not hysteria but rather of the nature of a paranoia in the broadest sense of the term. He also insisted on hereditary predisposition as an important etiological factor in such cases. It is impossible in a short review to mention the various analogous conditions which have been described and the opinions which have been expressed as to their nature, but anyone interested in the subject will find in this paper an excellent account of all that is known about this somewhat obscure but perfectly definite clinical state.

T. GRAINGER STEWART.

**BLADDER TROUBLES OF NERVOUS ORIGIN.** (Zum Kapital  
(416) der nervösen Blasenstörungen.) VOGEL, *Berl. klin. Woch.*,  
Oct. 23, 1905, p. 1372.

GENUINE bladder neuroses are usually betrayed by local discomfort and difficulty in initiating the act of micturition. They frequently have their origin in irritation at a distance. In disease of the spinal cord the amount of bladder disturbance is proportional to the extent of the lesion in the posterior columns. Absence of the ordinary sensations from bladder wall or from bladder neck and sphincter explains the difficulty. Neuroses are sometimes the consequence of toxic conditions of the blood and of the urine, as in the retention of coal gas poisoning, etc. One well recognised form is the irritable bladder, the so-called *bégaiement urinaire*.

S. A. K. WILSON.

**A STUDY OF THE AUTOMATIC PANDICULATION OF HEMI-  
(417) PLEGICS.** (*Etude sur la Pandiculation automatique des  
Hémiplégiques.*) BERTOLOTTI, *Revue Neurologique*, Oct. 15, 1905,  
p. 953.

ASSOCIATED movements are of three kinds, volitional, reflex, and automatic. In the first class are such functional and synergic acts as raising of the eyebrows, shutting of the eyes, etc., that we make when performing some other movement. In the second group of reflex synkinesias are movements consecutive to cutaneous excitation and due to the diffusion of the reflexogenous zone. Bilateral contraction of the cremaster, and bilateral plantar response from unilateral stimulation may be instanced. Finally, in a third category are movements associated with such involuntary spontaneous acts as yawning, sneezing, laughing, crying, etc. The general movement of extension of the limbs that accompanies yawning has been known since the time of the ancients by the name of *pandiculation*. The fact of its occurrence in infants, in a perfectly co-ordinated and symmetrical form, suggests that it is independent of the functioning of the pyramidal tracts. In hemiplegia the phenomenon manifests itself usually about the third month after the stroke, *i.e.* about the same time as the establishment of contracture. An examination of twenty-three cases of hemiplegia seems to indicate that it occurs only in those the function of whose pyramidal tracts is seriously interfered with. More particularly it is found in capsulo-ganglionic lesions, where the characteristic signs of a thalamic syndrome are present in hemianæsthesia, pain on the side of the paralysis, xerostomia, and disorders of swallowing and of micturition. Further, in most of the cases there existed a curious symptom which may be described as an attack of pruritus, occurring at intervals and affecting the limbs, or the root of the nose, or the scrotum, on the hemiplegic side. These turns of pruritus were often accompanied by involuntary extension of the forearm, with hyper-extension of the hand and fingers, and sometimes supination of the paralysed arm. The author is inclined to attribute to the optic thalamus an active automatic function in the motor innervation of different muscular groups.

S. A. K. WILSON.

**ON SLEEP'S ORIGIN. THE RELATIONS BETWEEN SLEEP  
(418) AND THE PITUITARY BODY'S FUNCTION.** (*Sull' origine  
del sonno. Studio delle relazioni fra il sonno e la funzione  
della glandula pituitaria.*) ALBERTO SALMON. Luigi Niccolai,  
Florence, 1905.

WHILE physiological sleep cannot be considered as an effect of a simple vasomotory or toxic process, it becomes perfectly com-



prehensible when its origin is ascribed to an internal physiological secretion.

The pituitary body, on account of its secretion, and inasmuch as it occupies moreover a special seat near psychic centres, and has a marked physio-pathological relationship with the central nervous system, is, then, the fittest gland for that very delicate function.

The existence of bromine in its glandular substance (Paderi) proves that it possesses hypnotic qualities on nervous centres, a hypothesis which is strengthened moreover by somnolence caused sometimes by the pituitary treatment and by its improving insomnia.

On examining sleep troubles in connection with lesions of the pituitary body, *somnolence* is observed in the following cases:—

1. In tumours of the pituitary body with or without acromegaly, characterised by glandular hypertrophy or without any degenerative features.

2. During the first phases of acromegaly, when a simple functional hyperactivity of the pituitary body is observed.

3. In myxoedema, which is almost constantly associated with hypertrophy of the pituitary body.

4. In sleeping sickness, in which hypertrophy of the pituitary body has been often ascertained; and in infections such as influenza, nona, etc., accompanied by meningitis, or slow evolving meningo-encephalitis, whose lesions constantly present congestive or inflammatory phenomena of the pituitary body (Wassiliw).

5. In some acute poisonings, especially if they increase the secretory phenomena, like that produced through a small quantity of pilocarpine, which increases every secretion, that of the pituitary body included (Guerrini).

6. In the slow auto-poisoning (hepatic, gastric intestinal, etc.) which experimentally produced a pituitary hypersecretion (Guerrini).

7. In obesity, the pathogenesis of which is often associated with functional pituitary body troubles.

8. In every diseased state capable of determining hyperæmia of the pituitary body and therefore its functional hyperactivity; for instance, congestion of blood in the brain, acute alcoholism, epilepsy in which the pituitary body was found greatly congested (Wenzel), head trauma, which not uncommonly is a cause of lesion of the pituitary body and of profound somnolence.

*Sleeplessness*, on the contrary, has been observed by the author in the following cases:—

1. In tumours of the pituitary body with or without acromegaly, accompanied by degenerative symptoms, and particularly in the state of acromegalic cachexy in which the gland is generally

destroyed by degenerative lesions; in malignant metastatic tumours.

2. In abscesses of the pituitary body.

3. In Graves' disease, the origin of which must be undoubtedly ascribed to a functional perversion of the pituitary body (Salmon), a gland which in some autopsies has been found extraordinarily atrophied (Benda).

4. In inanition or in old age during which one has observed the symptoms of functional insufficiency in the pituitary body, that is to say, a diminution of chromophile cells.

5. In the poisonings characterised by an inhibitory action on secretions, as in the case of atropine.

6. In diminution of the blood-pressure as in cardiopathy, and in neurasthenia, an affection whose special characters are precisely the diminution of arterial pressure and a general hyposecretion.

7. In the emotions generally manifested by secretory troubles.

Our hypothesis elucidates the disturbances of sleep found in affections of the internal secretory glands, all joined as they are by the closest physio-pathological relationship; we note, as an example, diabetes, an affection in which, besides the pancreas, other internal secretion glands are injured (thyroid, etc.), especially in some kinds of nervous diabetes of trauma, etc., in which it may be logically supposed that glycosuria is due to a poisoning of glycogenetic centres situated near the gland.

The relations between the pituitary body and the genital glands explain also sleep disturbances due to the modifications of sexual life, as the somnolence during pregnancy, after castration, generally accompanied by pituitary body's hypertrophy.

The anatomical and physio-pathological relations between nasal cavities and the pituitary body would finally explain many cases of narcolepsys and of insomnia produced by nasal troubles, and which were inexplicable till now.

These considerations allow me to venture upon the hypothesis that physiological sleep is essentially due to the internal secretion of the pituitary body, an hypothesis perfectly corresponding to the idea of the trophic anti-toxic action of the pituitary secretion on nervous centres.

AUTHOR'S ABSTRACT.

**A CASE OF ACROMEGALY WITH DESTRUCTION OF THE  
(419) PITUITARY GLAND BY HÆMORRHAGE** [Ein Fall von  
Akromegalie (Zerstörung der Hypophysis durch Blutung).]  
BLEIBTREU, *Munch. med. Woch.*, Bd. 52, No. 43, 1905, S. 2079.

THE author in this paper gives an account of his observations, clinical and post-mortem, in an interesting case of gigantism com-

bined with acromegaly. The patient, a young man of 21 years, first came under his observation in October 1904, when he complained of loss of strength, attacks of giddiness, headache, and sleeplessness. His previous health had been good, but at the age of seventeen his rate of growth rapidly increased until he attained a height of 1.96 metres. His head and extremities became very large, and his breadth of body also became proportionately increased. The nose, lips, and eyebrows were much thickened, and gave a mask-like appearance to the face; the voice was deep and harsh; the knee-jerks were very active, and there was no impairment of sensation. The gait was somewhat staggering, and Romberg's symptom was very marked. The fundus was normal. The urine was not increased in amount, and contained no albumin or sugar. The patient's intelligence was quite good, and with the exception of marked anæmia, nothing else of much clinical importance could be made out. The patient left hospital nine days later, feeling quite well, his gait being more certain, and Romberg's symptom having entirely disappeared. The diagnosis was gigantism with commencing acromegaly. In June 1905 he returned to hospital suffering from an attack of acute pulmonary tuberculosis, to which he quickly succumbed, his previous appearance having considerably changed owing to the rapid emaciation which now supervened, death occurring on July 10, 1905.

Post-mortem examination showed extensive pulmonary tuberculosis. The various organs (heart, liver, spleen, kidneys, and brain) were of very large size, but not more so than was to be expected from the condition of gigantism present. Microscopic examination of these organs revealed nothing abnormal. On removal of the brain no pituitary body could be seen, the infundibulum being rounded off at the end, and appearing as a sort of cone or peg-like structure at the base of the brain. The sella turcica was partially filled with a peculiar substance, the upper surface of which was rough and uneven and of a golden yellow colour. Further examination of this showed it to consist for the most part of blood pigment lying in a newly-formed framework of fibrous tissue, under which there still remained a thin layer of pituitary tissue in the form of a circle, arranged in alveoli lined by epithelium.

Such cases are of extreme rarity, and the occurrence of acromegaly and gigantism along with a destructive lesion of the pituitary tends to prove that these conditions are probably due to the absence rather than to increase or perversion of the secretion of the gland.

W. E. CARNEGIE DICKSON.

**PSYCHIATRY.**

**ON THE ÆTIOLOGY OF DEMENTIA PRÆCOX.** (Zur Aetiologie (420) der Dementia Præcox.) G. STEINER (of Ybbs), *Psych.-Neur Wchnschr.*, May 20, 1905.

STEINER refers shortly to the causes of dementia præcox which have been usually adduced. Out of 25 personal cases he found a syphilitic history in 4, and in 3 of these the psychosis followed immediately upon the syphilitic infection. He gives a brief abstract of each of the 4 cases. C. MACFIE CAMPBELL.

**CHRONIC MANIA AND PARANOIA.** (Chronische Manie und (421) Paranoia.) G. SPECHT (of Erlangen), *Centralbl. f. Nervenh. u. Psych.*, Aug. 1, 1905.

By chronic mania Specht denotes not a chronic condition developing out of acute or periodic mania, but a separate disease with characteristic course, which belongs to the constitutional psychopathic conditions, and, as a rule, develops at adolescence. Symptomatically it is a hypomanic condition, with occasional exacerbations, which may reach maniacal excitement or even delirious confusion: the prevailing mood is one of grumbling discontent. This mood inevitably leads to friction with the environment, and to the development of numerous delusions. A series of secondary symptoms thus develop round the hypomanic nucleus, and confuse the picture.

Cases of chronic mania are not rare, but are often diagnosed as paranoia, or are masked by chronic alcoholism. The features which suggest paranoia are the delusions of persecution, the self-assertive attitude of patient, his self-coined vocabulary of abuse, his peculiar mannerisms. In chronic mania, however, the delusions follow more variable anomalies of mood than are found in paranoia. They never become dissociated from a morbid emotional tone, which, according to Specht, is the fundamental change in paranoiac conditions.

The differential diagnosis is important from a medico-legal point of view: the paranoiac is more consistently dangerous than the patient with chronic mania. C. MACFIE CAMPBELL.

**THE FIELD OF DISTINCT VISION FOR FORM IN THE (422) INSANE AND THE BORN CRIMINAL.** (Il campo della visione distinta delle forme negli alienati e nei delinquenti-nati.) E. ANDENINO (of Turin), with 8 figures, *Archivio di Psich.*, Vol. xxvi., Fasc. i. ii.

THE author reports the results of his examination of 97 individuals with a perimeter, the ordinary runner of which was replaced by one which had a little window, at which one or two small black squares could be made to appear on a white background. The field of distinct vision for the form of an object is of course much more restricted than that for the mere presence of the object.

Out of 29 normal individuals, 3 showed retraction of the field of distinct vision for form. Out of 13 degenerates—criminals and individuals with moral insanity—who had normal fundi and acute vision, retraction of the field was observed in 8 cases. A similar proportion of adult epileptics showed the same feature, while in youthful epileptics it was less frequently met with. In other cases examined—hysteria, alcoholism, paranoia—retraction of the field was less frequently met with than among the epileptics and criminal degenerates. The peripheral organ was normal and retinal fatigue was eliminated. The retraction was most marked where the mental defect was greatest.

The author concludes that while peripheral conditions are of the first importance when an object is looked at directly, the perception of the form of an object not in the centre of the visual field demands a greater participation of cerebral functions, and is especially impaired where profound psychic anæsthesias exist.

C. MACFIE CAMPBELL.

### TREATMENT.

**EXOPHTHALMIC GOITRE AND ITS TREATMENT.** The Bradshaw (423) Lecture. GEORGE R. MURRAY, *Lancet*, Nov. 11, 1905, p. 1379.

THE author analyses 180 cases of exophthalmic goitre which have come under his personal observation. Females outnumbered males in the proportion of 17 to 1. In only 3 cases was there no enlargement of the thyroid gland at the time of examination and no history of previous enlargement. In 6 cases the left lobe was larger than the right. Exophthalmos was absent in more than 25 per cent. Tremor was only absent in 7 cases.

The author has not noticed the abnormal love of dress and craving for entertainment which have been described by Möbius. A "chorea of ideas" is an apt expression first used by Sir Russell Reynolds to designate a dominant mental characteristic of some of

these patients. Three patients of the series developed insanity, while in 10 in which there was no insanity definite hallucinations occurred. "In some cases convulsions occur which appear to be the direct result of the exophthalmic goitre." Gastric crises are occasionally observed.

As regards treatment in severe cases, confinement to bed is advisable, and large quantities of milk in addition to the ordinary meals should be given where there has been much loss of weight. A weak faradic current applied by means of two electrodes, one over the gland, the other over the back of the neck, for an hour or more twice a day, is, in the author's opinion, one of the most valuable methods of treatment. The electrodes, as suggested by Sir Victor Horsley, are fitted with straps and buckles at the sides by means of which they are fixed in position. They are then connected with the secondary circuit of a dry-cell faradic battery, a water rheostat being included in the circuit so that the strength of the current can be regulated as desired. The results obtained by Beck and Stegmann suggest that a further trial of an X-ray treatment should be made. In one of the author's cases treated in this way the symptoms seemed to be rather exaggerated.

Among drugs Dr Murray considers arsenic one of the most useful remedies for routine treatment. He recommends the addition of fifteen minims of tincture of convallaria in cases where the pulse-rate is over 100. Potassium bromide in 10 grain doses may be added to the mixture when nervousness is marked. Iodide of potassium and thyroid extract are harmful. Sodium phosphate has not yielded good results, but it is to be noted that he has only employed this drug in 15 to 20 grain doses.

Since 1895, when Enriquez introduced a new method of treatment, the serum of dethyroidised dogs, with beneficial results, a good deal of work has been undertaken on similar lines. Möbius' serum prepared from the blood of thyroidectomised goats is known commercially as antithyroidine. The author has used this serum to a limited extent without observing that it had any special effects. Various other serums are referred to. The administration of Rodagen, which is the dried milk of dethyroidised goats, has in some cases been beneficial and seems worthy of more extended trial. The author describes the case of a patient who suffered from peculiar convulsive attacks which he is inclined to attribute to the action of Rodagen, and he points out that this remedy should be used in doses not exceeding half a drachm.

The author has employed a serum which he prepared by feeding rabbits for a month or more on sheep thyroid extract. The rabbits are killed by bleeding and the blood collected with aseptic precautions in sterilised flasks. After standing for forty-eight hours the serum is decanted and 0.2 per cent of carbolic acid

added. A serum was also prepared from a goat in a similar way. In two cases of exophthalmic goitre in which this serum was employed, no special effect could be attributed to its use.

The risks of surgical treatment are so great that the author does not consider partial thyroidectomy advisable. He sums up his remarks on treatment in the following sentence: "We must on the whole conclude that at the present time, in the great majority of cases, the best results are obtained by general hygienic treatment combined with the use of electricity and certain drugs, and that as yet no serum or other animal product can be considered to give better results than these older methods of treatment."

EDWIN BRAMWELL.

**ANTITHYREOIDIN OF MOEBIUS IN A CASE OF BASEDOW'S (424) DISEASE WITH A PSYCHOSIS.** (*Antithyreoidin-Moebius bei Basedowscher Krankheit mit Psychose.*) G. LOMER (of Neustadt), *Münch. med. Wchnschr.*, May 2, 1905.

**A CONTRIBUTION TO THE SERUM TREATMENT OF (425) BASEDOW'S DISEASE.** (*Ein Beitrag zur Serumbehandlung des Morbus Basedowii.*) R. DÜRIG (of Munich), *Münch. med. Wchnschr.*, May 2, 1905.

**ON THE TREATMENT OF BASEDOW'S DISEASE WITH (426) ANTITHYREOIDIN OF MOEBIUS.** (*Zur Behandlung des Morbus Basedowii mit Antithyreoidin Möbius.*) A. ALEXANDER (of Berlin), *Münch. med. Wchnschr.*, July 18, 1905.

THE three authors report cases of exophthalmic goitre which were much benefited by treatment with antithyreoidin. In Lomer's case 110 c.cm. were given, the amount being distributed over eleven days. The pulse became slower, stronger, and more regular; the tremor and exophthalmos diminished; Graefe's sign disappeared; the goitre did not change. Two days after the drug was discontinued the pulse was as frequent as before administration and soon showed the same feebleness.

In Dürig's case the improvement was more definite and permanent, but 250 c.cm. were given, the maximum daily dose being 210 drops. Palpitation ceased; the pulse-rate diminished from 126 to between 75 and 85; tremor disappeared, as well as the signs of Moebius, Graefe, Stellwag; general nutrition improved. Half a year later the symptoms had not returned. An interesting feature during the treatment was the occurrence of a short period of apathy with a feeling of mental enfeeblement, which Dürig looked upon as a mildly myxoedematous condition. This fact

would tend to confirm the views of Möbius upon which the serum treatment is based.

Alexander reports three cases in which the serum treatment gave excellent results. The total amounts used were 43, 105, 40 grammes respectively. Body weight increased, the thyroid gland promptly became softer and smaller, tremor decreased, the exophthalmos disappeared in two cases, became less marked in the third. The beneficial action on the heart was no less marked. The author recommends that after 40-50 grammes of the serum be given the treatment be discontinued; should the symptoms recur, the patient is again to be submitted to a second period of treatment until 40 grammes more be given. By this method one avoids an overdose of the serum producing symptoms as in Dürig's case. Carbonic acid baths may with advantage be given during the treatment.

C. MACFIE CAMPBELL.

**ON THE TREATMENT OF NEURALGIAS.** (*Zur Therapie der* (427) *Neuralgien.*) SPITZMÜLLER, *Wiener med. Wochenschr.*, Sept. 30, 1905, p. 1922.

THE paper deals with the treatment of neuralgia by Schleich's cocaine infiltration. The writer refers to eleven cases, especially of sciatica, recorded by Lange in the *Münch. med. Wochenschr.*, No. 52, for 1904, as treated in this way, and records a most intractable case of his own which yielded to this method. The latter was that of a lady, aged 32, suffering from neuralgia almost constantly in all three branches of one or other trigeminal nerve. She had suffered many years, and among other remedies had tried colchicum, gelsemium, phenacetin, caffeine, morphia, chloroform, menthol, aspirin, trional, chloral, cannabis indica, hyoscyamus, pyramidon, mesotan, antipyrin, dionin, bitter almond water, veronal, bromidia, salochinin, benzoate of soda, salipyrin, citrophén, electricity, diet cures, tooth extraction, nasal treatment, hypnotism, etc., etc. During an intense attack of pain the writer injected a solution of cocaine and suprarenin into the supra-orbital nerve, with instant relief in the upper part of the face. This he followed by injections at the point of exit of the infra-orbital, mental, and occipital nerves, with complete cessation of pain. After about twelve hours the neuralgia returned and the injection was repeated. The process was repeated nine times, after which the patient, at the time of writing, had remained free of neuralgia for six and a half months, save for occasional warnings. The writer gives directions as to the technique of the injection.

JOHN D. COMRIE.



**THE VIENNA THERAPEUTIC EXPERIMENTS IN GENERAL**(428) **PARALYSIS.** (*Die Wiener Heilversuche an Paralytikern.*)K. ALT, *Psych.-Neur. Wchnschr.*, April 8, 1905.

THIS is a short statement and criticism of the results arrived at by Pilcz, who, on Wagner's inspiration, treated a series of general paralytics with Koch's tuberculin until a febrile reaction was obtained. The method was suggested by the observation that an acute fever may lead to marked improvement in chronic psychoses.

Pilcz found, on comparison of 66 cases treated in this way with other cases not so treated, that the progress of the disease seemed delayed by the treatment, and that those patients who survived were in better health than other patients who had not received the tuberculin.

C. MACFIE CAMPBELL.

**CONSIDERATIONS ON THE OPERATIVE TREATMENT OF**(429) **SPASMODIC TORTICOLLIS.** (*Einige Reflexionen über die operative Behandlung des Torticollis spasticus.*) JOHN BERG,*Nord. med. Arkiv.*, 1905, Afd. 1, H. 1.

THE author describes five cases operated upon; three were successful to such an extent that the patients were able to undertake regular work of which they were previously incapable, but none were quite free from convulsive twitchings. In all there were complex spasms affecting the accessory nerve of one side and the deep muscles of the back of the neck on the other, and in one case at least, the face and arm were also affected.

The surgical treatment varied, but in the main involved both resection of the spinal accessory nerve and cervical neurectomy. But at the same time the suggestive influence of the operation could not be excluded; as when, for example, extensive spasms disappeared immediately after simple division of the spinal accessory.

In all, indeed, active suggestive treatment appears to have played a considerable part in the cure.

In his general consideration the question asked is, "whether surgical treatment is justifiable, and if so, on what grounds and according to what principles?" The position he takes up is—that so long as suggestive treatment, especially Brissaud's re-educative method, has not been first thoroughly tried, any operative treatment is unjustifiable. But since suggestive methods are not always successful, surgical interference may be justified although the indications for it cannot yet be definitely laid down. The prospects of success from operation are better, the more circumscribed and fixed is the spasm, and the less the psychical element

present. As regards choice of operation, Kocher's multiple muscle division has the disadvantage of requiring repeated operations, and since the results obtained, good though they may be, appear to be due to non-union of the divided nerves, he prefers a method of nerve resection. The spinal accessory should only be divided if the sterno-mastoid is markedly involved. The hinder branches of the upper cervical nerves supplying the strong deep rotators usually require division, but stress is laid on the difficulty of finding these branches.

J. H. HARVEY PIRIE.

**THE ESTABLISHMENT OF CEREBRAL HERNIA AS A DE-  
(430) COMPRESSIVE MEASURE FOR INACCESSIBLE BRAIN  
TUMOURS.** HARVEY CUSHING, *Surgery, Gynaecology, and Ob-  
stetrics*, Vol. i., No. 4, 1905.

IN discussing the symptoms of brain tumours, Dr Cushing states his opinion that blindness is in all probability the immediate result of venous stasis, and not of an inflammatory process such as the term optic neuritis would imply. He tells of relief to symptoms of intra-cranial tension brought about by spontaneous herniation of the brain, or by separation of the sutures, the latter occurring in the case of three children aged 4, 5, and 8 respectively.

The writer suggests that in cases where there are no certain signs by which the growth can be localised, or in cases where the tumour is manifestly in a part of the brain without the reach of removal, that a "silent area" of the brain should be selected over which to trephine, so as to limit, as far as may be possible, the paralysis which is sure to follow the herniation of parts in the motor area.

In order that the "decompressing" action of the hernia may be effectual, it is necessary not only to remove bone, but also the dura mater, over a considerable area.

He describes a method of removing bone and dura in the temporal fossa, under the temporal muscle, so as to allow of herniation of the brain in the neighbourhood of the temporo-sphenoidal lobe, the split temporal muscle being brought together over the hernia, and serving both to limit its size and to act as a protection. This operation he has carried out successfully in certain cases on both sides of the head.

This method Dr Cushing calls the "Temporal *intermuscular* method." One would suggest that any one of the terms, "sub-muscular," "infra-muscular," or "intra-muscular" might be more appropriate.

A plan is also given for performing a similar decompressive operation in the occipital region in cases of cerebellar compression.

Dr Cushing's experience has been encouraging, and, like many other surgeons, he has frequently given great relief in very distressing cases.

J. M. COTTERILL.

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## Reviews

**PSYCHIATRY: A TEXT-BOOK FOR STUDENTS AND PHYSICIANS.** STEWART PATON. Philadelphia and London, J. B. Lippincott Company, 1905, pp. 618.

A GOOD text-book of psychiatry in the English language would at the present moment be of the greatest service to the student, for in none of the existing books is there a presentation of the subject which takes adequate account of those modern conceptions which have altered to such an extent the clinical grouping of cases of mental disease. There is still a tendency to cling to the old symptomatological traditions, and a shyness of adopting a psychiatry made in Germany.

Paton does not profess to furnish an exhaustive treatise; he has aimed at giving the results of modern clinical and experimental investigations, and "at presenting the different views of leading authorities in a manner readily to be comprehended by students." From both points of view it must be admitted that the book is rather disappointing; the author has shaken himself free from older traditions only to embrace the dogmas of a more modern school, and the views expressed seem based less upon a critical digest of personally observed cases than upon a familiarity with recent literature. There is a tendency throughout to adopt an abstract and frequently involved form of statement, and to refer to hypotheses of doubtful value. This general tendency is in part corrected by the introduction of histories of cases to illustrate the various psychoses.

In the first 224 pages, Paton discusses the various disorders met with in mental disease, and general questions such as treatment and ætiology. Chapter IV. should be of distinct value to the student, as it describes in a clear and well-arranged manner the method of examination of patients. In the second part the author passes to the clinical grouping of mental diseases, and to the detailed description of the various psychoses, and here the influence of Kraepelin is predominant. The descriptions of the various psychoses as a rule are adequate, but one misses a statement of the larger issues and of the problems that await solution; thus the three forms of dementia præcox, hebephrenic, catatonic,

and paranoic are described without any sufficient discussion of the grounds for the establishment of this much disputed clinical group, and the author discusses the ætiology of dementia præcox as a whole as if it were one definite disease. Bonhöffer's case, quoted in the foot-note to p. 405, was a catatonic syndrome relieved by operation, and not due to operation, as stated in the text.

Paton adopts Kraepelin's large group of manic-depressive insanity in which are swamped the old simple mania and melancholia as well as the recurrent forms; but while it must be granted that what would previously have been called simple mania or melancholia is frequently only one phase of a polymorph disease, there are cases where there seems to be no tendency to a recurrence of the same or of the opposite type of attack. The analysis of such cases is of great importance, for after all we do not know what the meaning of the manic syndrome is, and whether these simple cases may not essentially differ from cases with a tendency to recur. The manic complex may occur in other psychoses, *e.g.* dementia paralytica.

The author's description of the symptomatology is rather misleading; choreiform and ataxic movements are not a good expression for the exuberant activity of the manic, while the reckless self-injury described belongs to the maniacal excitement of some other psychosis. If the student refer to the general discussion in Chapter iii. for a sample of the manic flight of ideas, he will find a long and misleading one where the flighty associations are hard to find, and not the prominent feature; yet it would have been so easy to give a typical example in a few lines.

In discussing paranoia, Paton refers to the views of several authors. "Berze refers the primary disturbance in paranoia neither to an intellectual nor to an emotional state, but to an anomaly in apperception of such a character that the process of bringing the psychic content into the field of consciousness is impaired. This anomaly of function results in the impairment of the apperception, and upon this derangement depends the lack of judgment and defective critical power of the paranoic." The bewildered student will have some difficulty in seeing how he is to find out whether Berze is right or wrong.

It would take too much space to discuss the author's descriptions of the various psychoses in detail; the above remarks may serve to show the weak points and the good points of the book. It is true that it lacks individuality, is frequently rather obscure in statement, and refers rather exclusively to the periodical literature of the last five years; on the other hand, old rubrics have been discarded, a much more thorough and systematic

examination of patients shown to be necessary, the general classification adopted is practical and an excellent starting-point for investigation, while the descriptions of the various psychoses are supplemented by the complete records of actual cases.

C. MACFIE CAMPBELL.

**LEHRBUCH DER NERVENKRANKHEITEN FÜR ÄRZTE UND STUDIERENDE.** Prof. Dr H. OPPENHEIM, Berlin. Fourth Edition. S. Karger, Berlin, 1905. 30 M.; geb. 33 M.

PROFESSOR OPPENHEIM'S text-book of nervous diseases first appeared in 1894. Editions No. 2 and 3 followed in rapid succession, each marked by a steady increase in size and in the number and excellence of illustrations. Now we have Edition 4, containing over 200 extra pages and 24 new illustrations, and published in two volumes—a feature which adds greatly to the facility of handling it. This increase of subject-matter is effected by the working up of each section, the whole range of encyclopedic contents being brought fully up to date, arranged with great method and conciseness, and set forth in language so clear and precise that the book is one particularly easy to read and to consult. Although space did not permit of the addition of bibliographies to each subject, all the recent developments of value have been included; such chapters as those on hysteria and neurasthenia—subjects affording so wide a field for discussion—have been largely re-written, and as a result, Oppenheim's work is perhaps the most complete and useful text-book on nervous diseases in the German, or for that matter, in any language. Amongst the illustrations added to this edition there is a valuable plate showing the innervation of the sphincters.

We are glad to see that in his preface Oppenheim lays stress upon the urgent need for special training in neurology, and for its treatment as a science quite independent of general medicine on the one hand, or of psychiatry on the other.

ALEXANDER BRUCE.

**ISOLEMENT ET PSYCHOTHÉRAPIE. (TRAITEMENT DE L'HYSTÉRIE ET DE LA NEURASTHÉNIE.) PRATIQUE DE LA RÉÉDUCATION MORALE ET PHYSIQUE.** Par les Drs JEAN CAMUS et PH. PAGNIEZ, Anciens Internes de la Salpêtrière. Préface de M. le Professeur DÉJÉRINE, Médecin de la Salpêtrière. Felix Alcan, Paris, 9 fr.

THERE is no more remarkable illustration of recent progress in therapeutics than the altered attitude of the profession to the

psycho-neuroses. It is fitting that this monograph, which will be read with the greatest interest and profit by all who have to do with the treatment of hysteria or neurasthenia, whether in hospital or private practice, should emanate from the School of Paris, whose great masters have done so much to elucidate the intricate problems which these functional disorders present.

The book consists of three parts—firstly, an account of the various methods formerly employed and at present used in treating these maladies; secondly, an elaborate description of the measures advocated by the authors; and, thirdly, records of a large number of cases.

Persuasion, by which is implied a direct effect produced by appealing to the patient's reason and his will, has replaced those practices enshrouded with mystery which formerly occupied the field. Isolation *per se* is to be regarded as merely an adjuvant which permits of the application of psycho-therapeutics under the most advantageous circumstances.

A striking feature of the work is the manner in which explicit directions are set forth in relation to every point likely to arise during the course of treatment, and the way in which possible difficulties are to be combated. The book is so written that the reader can, for the time being, picture himself in the position of the patient.

Even the physician who has had a large experience in dealing with the psycho-neuroses will learn much by a perusal of these pages.

EDWIN BRAMWELL.

**ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUT AN DER  
WIENER UNIVERSITÄT.** Herausgegeben von Prof. OBER-  
STEINER. H. x., 1903. Deuticke, Vienna. Price 25s.

THIS annual publication has become a necessary possession of every well-equipped Neurological Library. The tenth volume fully maintains the high standard of its predecessors. For further details regarding the contents of the volume, the reader is referred to abstracts of papers which it contains, which have appeared in recent numbers of the *Review of Neurology and Psychiatry*.

1. J. Karplus. "Family Likenesses affecting the Fissures of the Human Cerebrum" (abst. A. W. Campbell, p. 520).
2. Okada. "Experimental Observations on the Vascular Nutrition of Peripheral Nerves" (abst. Purves Stewart, p. 520).
3. Obersteiner. "Action of Radium Rays on the Central Nervous System" (abst. P. T. Herring, p. 534).

4. Okada. "Action of Radium Rays on Muscles and Peripheral Nerves" (abst. P. T. Herring, p. 535).
5. Redlich. "On the Comparative Anatomy of the Association System of the Mammalian Brain" (abst. Gordon Holmes, p. 603).
6. Zuckerkandl. "The 'Affenspalte' and the Occipital Operculum in the Human Brain" (abst. Elliot Smith, p. 729).
7. Modena. "Degeneration and Regeneration of Peripheral Nerves" (abst. R. A. Fleming, p. 614.)
8. Bier. "The Anatomy of the Central Nervous System in a Teratological Specimen" (abst. David Waterston, p. 536).
9. Neurath. "Anatomy of Poliomyelitis Anterior Acuta" (abst. F. E. Batten, p. 528).

EDWIN BRAMWELL.

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